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A FEW OBSERVATIONS ON THE HAEMODYNAMICS OF THE NORMAL CIRCULATION; AND THE CHANGES WHICH OCCUR IN AORTIC INSUFFICIENCY.[1,2]

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The phenomena associated with the physiology of the normal circulation and of the circulation as it is modified by various pathological processes are so numerous and so complicated that they have formed the subject of much experimental and theoretical study. The student of these phenomena finds in the literature a vast store of experimental data, some of which seems difficult to understand in the light of our present conceptions, and a wealth of theoretical observations, partly contradictory and of necessity partly incorrect.

The following paper presents a study of some of these phenomena in the light of a few principles of hydrodynamics.

Hydrodynamics is the science which deals with the behavior of fluids in motion; and that part especially which discusses the forces concerned in the flow of water through pipes will help us somewhat in our study of the forces concerned in the flow of blood through the arteries, realizing always that the conditions are comparable but by no means identical, and that the differences may necessitate considerable modification of our conclusions. We propose to introduce straightway a few principles of hydrodynamics, then to apply these principles to normal arterial flow and finally to discuss the haemodynamics of aortic insufficiency. The reason for discussing aortic insufficiency is that in this condition the circulatory changes are markedly exaggerated and help us understand the conditions which obtain in the normal circulation.

For our purposes, the most important law connected with fluids in motion is that, when water flows through a pipe, the total amount of

[1] I wish to thank Dr. Edward P. Carter for his encouragement and guidance, and Dr. William H. Howell for reading and criticizing the manuscript.

[2] Submitted for publication, July 31, 1928.

energy possessed by a unit mass of water passing a given point is the sum of three components; first, the pressure head (side pressure) which is measured by the height to which fluid will rise in a vertical tube (piezometer tube) placed in the fluid at that point; secondly, the velocity head, or kinetic energy possessed by a unit mass of water by virtue of its forward motion and its tendency to continue in that motion,—in other words, if the motion is stopped, energy will be released or trans-

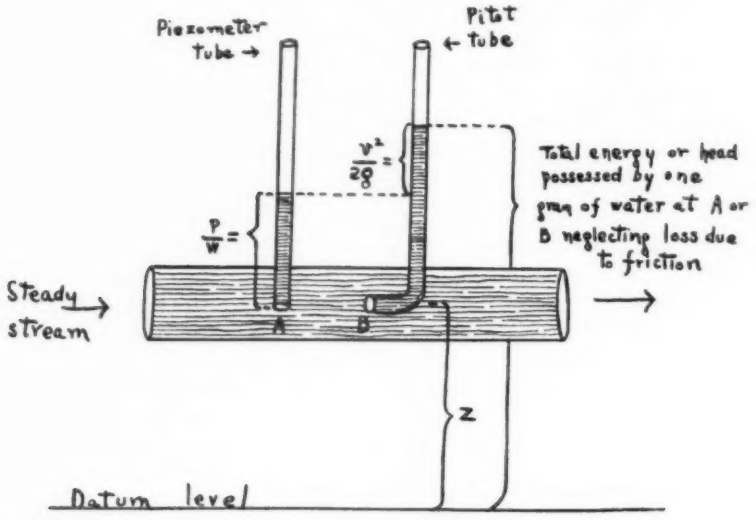

FIG. 1. DIAGRAM TO ILLUSTRATE THE THREE COMPONENT FACTORS OF THE TOTAL ENERGY POSSESSED BY A UNIT MASS OF WATER FLOWING THROUGH A PIPE

formed; thirdly, the elevation above the datum level, *i.e.*, a unit of mass which is 100 feet above the ground will have energy because of its position, this energy being transformed steadily into kinetic energy as it falls to the ground.

These components may be written as follows:

$$E = \underset{(1)}{\frac{p}{w}} + \underset{(2)}{\frac{v^2}{2g}} + \underset{(3)}{z}$$

E equals the total energy possessed by a unit mass, *i.e.*, 1 gram, of water passing through the pipe at any given point.

Expression (1) indicates the height (h) to which the fluid will rise in the piezometer tube, measures the pressure head, and is equal to p, the pressure in grams per square centimeter, divided by the weight of 1 cc. of fluid; expression (2) represents the kinetic energy or velocity head and is equal to the height to which one gram of water would rise if it were directed upward by its kinetic energy, this height in centimeters being equal to $V^2/2g$, when v equals the velocity of the fluid expressed in centimeters per second, and g equals the force of gravity in dynes or c.980; expression (3) represents the energy due to elevation above the datum level and is expressed in centimeters.

If the patient or animal be kept in the horizontal position, there is very little change of level of the blood in its course through the body, and since z changes so little, it may be neglected.

Consider a branched pipe shaped as in figure 2 and lying horizontally with b′ and c′ at the same level, a steady stream of water being forced in at A and escaping at b′ and c′. It is apparent that the velocity of flow in the above system is greatest where the cross-section area is least, *i.e.*, at A. If there is no loss of energy due to friction, and if *Z* does not change, the pressure head must increase as the velocity head decreases, acording to Bernouilli's Theorem—"In a steady moving stream of an incompressible fluid in which the particles of fluid are moving in stream lines, and there is no loss by friction or other causes

$$\underset{\text{pressure head}}{\frac{p}{w}} + \underset{\text{velocity head}}{\frac{v^2}{2g}} + \underset{\text{elevation above datum level}}{z}$$

is constant for all sections of the stream" (1).

Obviously the velocity decreases as the water passes from B to C; hence the pressure head increases. If the sum of the cross-section areas at E and E′ is greater than the area at C or D, the velocity at E or E′ will be less than the velocity at C or D, and conseqently the pressure head at E or E′ will be greater than the pressure head at C or D. When the tube is greatly constricted, the velocity head is greatly increased, and the pressure head may be reduced below atmospheric

pressure as occurs in the ordinary suction pump that we attach to the faucet.

Figure 3 is a familiar diagram from Tigerstedt to illustrate the hydrodynamics of the circulation. Neglecting loss of head due to friction or other causes, the total head at A, the bottom of the reservoir, is equal to the height H plus atmospheric pressure. At E where the water is

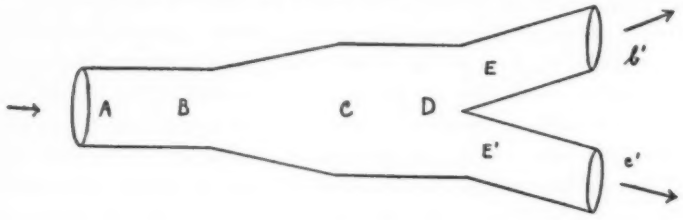

FIG. 2

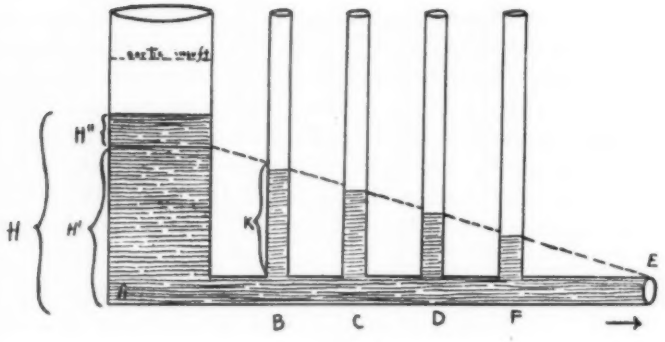

FIG. 3

escaping, the total head equals the velocity head plus the atmospheric pressure. In other words, the pressure head at A due to the height of the water H is completely transformed into velocity head at E. Under such circumstances the pressure head along the pipe from B to E is equal to atmospheric and there will be no tendency for water to rise in the piezometer tubes B, C, D and F.

Under ordinary conditions, however, there is a variable loss of head

due to friction along the pipe AE. Neglecting other causes for loss of head,

Total Head at A = total head at E + Loss of Head due to friction.

There is, therefore, a gradual loss of head as the water proceeds from A to E. Since the water is flowing steadily, the velocity head is constant all along the pipe. We may conclude, therefore, that the pressure head falls steadily from B to E, and this conclusion is born out by the pressure gradient expressed by the varying height to which the water will rise in the piezometer tubes at B,C,D and F. The pressure head K at any point B is determined by and varies with the resistance which has to be overcome, between the point B and the point of exit E.

If the opening at E is closed gradually (to avoid water hammer), the flow of water will be stopped, the velocity head will be converted into pressure head, and the total head will be equal to the pressure head, *i.e.*, the fluid will rise in all the tubes to the level of the fluid in the reservoir.

It can be seen from the diagram that if H represents the total head (above atmospheric) H′ represents that portion of the total head which is expended in overcoming resistance and H″ represents that portion of the total head which is converted into velocity head and which determines the velocity of the escaping fluid.

In the normal circulation, the main source of energy is the heart muscle, and the pressure within the ventricle during contraction is the source of the pressure head and velocity head within the aorta. In any case, (neglecting loss of head at the aortic orifice due to expulsion of blood through a small orifice), the velocity head within the aorta will be equal to the total pressure head within the ventricle, diminished by the loss of the head due to friction or other causes. The pressure head within the aorta will be equal to this same loss of head due to friction or other causes. The arteries going to the upper extremity and head arise at right angles from the arch of the aorta, and form very convenient piezometer tubes for obtaining the pressure head in the aorta.

DYNAMICS OF CARDIAC ACTION

If a small weight be suspended from a strip of striated muscle which is then stimulated to contract, the muscle will shorten and lift the

weight. From a study of kymographic curves it may be seen that the weight, once it starts to rise, ascends with uniform velocity, *i.e.*, its acceleration is zero. We may conclude, therefore, that during the ascent of the weight, *i.e.*, during the period of muscular shortening, the force of the muscular contraction is exactly equal to the force which gravity exerts upon the suspended weight. Since the latter force is constant, the former must be constant. While the muscle is shortening, its contraction is isotonic, *i.e.*, its force of contraction is constant.

If the suspended load is considerably increased so that it cannot be lifted at all, the contraction of the muscle will be isometric, its length will remain constant, but the lifting force which the muscle exerts upon the weight will be steadily increased during the period of isometric contraction. It is conceivable, therefore, that the load may fall intermediately between these two extremes, with the result that the early part of the contraction is isometric, and is characterized by an increase of contracting force without shortening, while the latter part of contraction begins as soon as the lifting force becomes slightly greater than the force of gravity acting upon the load, and the period of isotonic contraction begins, characterized by actual shortening, the lifting force remaining constant. These, indeed, are the conditions which obtain in the normal cardiac contraction. In the earliest part of ventricular systole, the contraction is isometric, *i.e.*, the size of the ventricular cavity is not reduced until the intraventricular pressure is sufficiently high to force open the aortic leaflets, permit the escape of blood, and allow the ventricular muscle to contract isotonically. As will be seen directly, the actual load decreases as ventricular contraction advances.

This conception of muscular contraction suggests that every contraction may be divided into two phases, an early isometric period, followed by a later isotonic period, and that the length of the isometric period, other things being constant, depends upon the size of the load to be lifted. It is quite conceivable that the so-called latent period of muscle contraction is an expression of the isometric period during which the force is increased sufficiently to accelerate the load upward, and that this period is so very short (0.004 second) merely because the load is small and the muscle comparatively strong.

For purposes of general discussion the ventricle may be considered a

hollow sphere which forces its contents through the aortic opening whenever the size of the cavity is reduced by the shortening of the fibers which form the wall of the cavity or surface of the sphere. The muscle fibers may be considered as meridians of the sphere, lying along the circumference of great circles.

After these fibers have been stimulated, they jointly exercise a compressing force on the contained blood, and when the isometric period is terminated, they actually shorten, and diminish the size of the cavity, as well as the surface of contact between the blood and the ventricular wall.

At any particular instant the force of contraction of the muscle wall is transmitted to the contained blood which is in contact with the inner surface of the ventricular wall. The pressure head which is developed equals the total force divided by the area of the inner surface of the wall. During the isometric period, the contracting force increases, while the surface area remains constant, and consequently the pressure head rises.

During the period of ejection the surface area, which is proportional to the square of the radius of the sphere, diminishes very rapidly. If the contraction of the muscle fibers during the period of ejection is really isotonic, then the total force remains constant, and since the area rapidly decreases, the pressure head must rise during this period. It is known of course that the pressure head in both the ventricle and aorta rises during systole. The force of ejection at any instant depends upon the excess of intra-ventricular over intra-aortic pressure. As the pressure head in the aorta rises, the load on the heat muscle increases. At the same time, the surface area of the ventricle is decreasing, and with the same force of muscular contraction the ventricular wall can develop a much higher pressure head.

The decrease in surface of the ventricular wall, neglecting for a moment the rise of intra-aortic pressure during systolic ejection, is tantamount to decreasing the load on the cardiac muscle, because it can now maintain the same pressure head with less contractile force. We shall be able to understand these changes more completely when we have studied the effect of gradual diminution of the load upon the force and rapidity of a contracting muscle. It seems *a priori* that the diminution of the load will not arrest the intra-muscular changes that make

for contraction, once these changes have been started, and that, the muscles will contract just as forcibly and perhaps more rapidly. This, however, is still to be determined.

It should be noted that the diminution of the load on the cardiac musculature which takes place during the period of ejection, and is due to the decrease of surface area, is opposed by the increase of the load which takes place during the same period and which is due to the increase of intra-aortic pressure. What the net effect of these opposing forces may be can only be determined after more detailed study. It is obvious, however, that the rise of intra-aortic pressure head during the ejection period depends upon the energy of ventricular contraction and that this rise being derived from systolic ejection tends to oppose further ejection of the blood.

If we assume that during the period of ejection the cardiac muscle fibers shorten at a uniform rate, that is, that the amount of shortening per unit of time is constant, we are led to some interesting conclusions. Considering spheres of different size, the circumference of a great circle is proportional to the radius of the sphere, the surface area of the sphere is proportional to the square of the radius, while the volume of the sphere is proportional to the cube of the radius. The muscle fibers are distributed along the lines of great circles and their length is proportional to the radius of the sphere. The surface of contact between the blood and heart wall corresponds to the surface of the sphere. The capacity of the ventricular chamber corresponds to the volume of the sphere. If the length of the muscle fiber decreases with uniform velocity the surface area will decrease with the square of that velocity, while the volume will decrease with the cube of that velocity. If the total force of muscular effort remains constant during the period of ejection, the pressure head in the ventricle, being equal to that total force divided by the surface area, will rise very rapidly because of the very rapid decrease in surface area. Since the radius of a sphere is proportional to the circumference of a great circle, a decrease of length of the muscle fiber with constant velocity is accompanied by a similar decrease in the length of the radius. Since the volume of a sphere is proportional to the cube of its radius, it follows that during the time that the radius is reduced to one-half its former value, the volume will be reduced to one-eighth of its former value. The obvious conclusion is, therefore,

that if during the period of ejection the heart muscle shortens at a uniform rate, the velocity of ejection will be far greater at the earlier than at the later part of that period. If the force of contraction is constant, the pressure head will be much higher at the later part of the period of muscle-shortening than at the earlier part of that period.

Such considerations help us to understand the physical principles underlying the intra-ventricular and intra-aortic pressure curves which Wiggers has been able to obtain. It must be borne in mind that pressure changes which occur so rapidly are difficult to measure with any great degree of accuracy. The cannula, too, must be so inserted into the cavity that the plane of its orifice will lie parallel to the direction of flow of the blood, otherwise the readings which are supposed to record changes in pressure head will be influenced by velocity head, *i.e.*, they will be increased if the cannula points against the stream, decreased if the cannula points with the stream.

WORK OF THE HEART

Current ideas with regard to the work of the heart may be obtained from Howell's brief discussion (p. 555) and calculations, based upon figures from Tigerstedt (2).

The work of the heart is calculated by considering the column of blood which is pushed out against a pressure of 15.0 cm. of Hg. The result given is 19,950 gm.-cm. of work.

The kinetic energy of the ejected blood is calculated by the formula, realizing that the ejected blood has been accelerated from a velocity of zero in the ventricle to an average velocity of 50 cm. per second in the aorta, and found to be 135 gm.-cm. It is pointed out that the velocity factor comprises less than 1 per cent of the total amount of work done by the ventricle. This method of calculation is not above criticism.

In the first place the ventricular contents are not ejected against a pressure of 15.0 of Hg. That may represent the maximal systolic pressure in the aorta, but, as we have seen above, most of the blood is ejected during the early part of systole, when the pressure in the aorta is at its diastolic level, and the rise of pressure in the aorta depends upon the blood which has already been ejected from the ventricle. Instead of using the systolic level, therefore, in calculating the work of

the heart, we ought to take a value nearer the diastolic level, because most of the blood is discharged during the earlier part of the period of ejection. Since all our figures are approximations, at best, we may say that according to this method the work done by the heart in transferring the blood from the ventricular to the aortic cavity is nearer 12,000 gm.-cm. than 19,950.

Assuming that the ejected blood after reaching the aorta is moving with an average velocity of 50 cm. per second, the kinetic energy possessed by the ejected blood is calculated to be 135 gm.-cm., which still gives us about one per cent.

The fallacy of this latter comparison arises from ignoring the fact that the velocity head of the whole mass of arterial blood has been greatly increased. If the average velocity of arterial flow is 50 cm. per second, the systolic velocity must be considerably above that and the diastolic considerably below. Merely for the sake of comparison, let us assume that the highest velocity is 75 cm. per second and the lowest 25 cm. per second. It would follow that cardiac contraction has succeeded in accelerating the *whole mass* of arterial blood from its diastolic to its systolic velocity, and that the increase in velocity head of the whole mass of blood is derived from the energy of ventricular contraction, for there is no other source. If we assume that the mass of arterial blood which has received this increase of velocity head is about 1000 cc., the increase in velocity head will be sufficient to increase the kinetic energy of the arterial blood by about 1350 gm.-cm. The figures offered are merely conjectural, and their value lies merely in this, that they indicate that heretofore we have underestimated the portion of cardiac work which is translated into kinetic energy during systolic contraction. A large portion of the work of the heart is translated into potential energy of the arterial walls which are stretched during systolic distention.

The influence of respiratory movements upon cardiodynamics is not without interest. The mechanics of inspiration consists in an increase in the size of the thoracic cavity with a consequent decrease of pressure. The air rushes in to fill the lungs, which expand because of the diminished pressure in the space which lies between them and the chest wall. It is of significance to us that the heart lies in that space. During cardiac diastole the pressure exerted on the blood in the ventricle,

assuming for a moment complete ventricular relaxation, is equal to the pressure in the mediastinal space. During systole, there is an increment due to ventricular contraction. In terms of absolute pressure, the first component equals about 760 mm. of Hg. while the increment due to ventricular contraction equals about 150 mm. of Hg. When the pressure is measured with a mercury manometer it will be found to be 150 mm. of Hg. *i.e.*, 150 mm. above atmospheric pressure.

During normal inspiration the pressure in the mediastinal space may be reduced by about 8 mm. of Hg., *i.e.*, to 752 mm. of absolute pressure. Assuming the increment due to ventricular contraction to be unaltered, *i.e.*, 150 mm. of Hg., it follows that when the pressure is now measured, it will be found to be 142 mm. of Hg., *i.e.*, 142 mm. above atmospheric.

In 1915, Erlanger from his experimental studies concluded (3) that systolic pressure falls with inspiration and rises with expiration. It has been variously explained as due to change in heart rate, asssociated activity of nerve centers in the medulla, *etc.* Erlanger does not offer any explanation except that he is inclined to believe that "the cause will be found to lie mainly, as Sahl has surmised, in the changing capacity of the blood vessels of the lungs."

While determining the brachial blood pressure in a patient with bronchial asthma, we found the reading to be about 135 systolic, over 100 diastolic. As the height of the mercury reached 135 mm., the sounds in the ante-cubital fossa became audible, but came through in groups of three, followed by a period of silence which we soon noticed to be synchronous with inspiration. This inspiratory period of silence persisted even at the diastolic level of 100 mm. of Hg. The interpretation, as we see it, is that respiratory pressure-changes in the mediastinum are grossly exaggerated in cases of bronchial asthma, which is to be anticipated from the increased effort of respiratory movements. The constricted bronchioles obstruct the inflow of air during inspiration, and the increased inspiratory effort to overcome this obstruction greatly reduces the pressure in the mediastinal space. In the patient referred to above, the inspiratory fall of systolic pressure was at least 35 mm. of mercury, and may be taken as a measure of the extraordinary reduction of mediastinal pressure which occurs in an asthmatic during inspiration.

Erlanger found that the respiratory variation of blood pressure in the normal man were about 7 or 8 mm. of Hg.; it will be noticed that this change of pressure is just about equal to the changes of pressure which occur in the mediastinum during normal respiration.

DYNAMICS OF ARTERIAL FLOW

In 1906, Percy M. Dawson reported the results of his studies on *The Lateral Blood Pressures at Different Points of the Arterial Tree* (4). Dawson's method was to take manometer readings of the blood pressure in many of the larger arteries, valved manometers being used to determine maximum and minimum pressures, and determining the mean pressure by constricting the communication to the mercury column and thus decreasing in amplitude the excursions of the mercury column which correspond to the heart beat. He took maximum, minimum, and mean pressure readings from many arteries and many dogs. In order to permit comparison of the findings obtained from different animals he reduced each pressure reading to the reading one might be expected to find in the normal average, ideal dog.

In his determination of *Lateral pressures* at different points in the arterial system Dawson has measured the variations in *pressure head*, which of course is merely another name for the same energy. In choosing the femoral artery as a basis of comparison for reducing his observed readings to values for the average or "schematic" dog, Dawson really made an unfavorable choice. What he was attempting to measure and compare was pressure head, maximal, minimal, and mean, at different points. These values are correctly given by connecting a mercury column to arteries which leave at right angles from the main trunk at the point where the pressure head is to be measured. It would be better to have chosen as a standard some artery that measures only pressure head, *e.g.*, the left subclavian rather than the femoral artery, where the mercury manometer reading indicates the sum of pressure head and velocity head, and such increment as may result from the effect of water hammer. The maximal systolic pressure in the left femoral artery is given as 188 mm. of Hg., while the maximal pressure in the deep femoral is 152 mm. of Hg. The calculated differences of 36 mm. of Hg., indicating the excess due to velocity head plus water hammer, may even be greater if the deep femoral

comes off at an acute rather than at a right angle, for in the former case its maximal reading (152 mm. of Hg.) is increased by such part of the velocity head (and water hammer) which it derives from the main vessel.

The objection to the use of the femoral standard in comparing values for arterial pressure head lies in this, that there may be considerable variations in velocity head (and effect of water hammer), although the pressure head in the unobstructed system is only slightly increased. A standard which varies widely because of factors which have very little effect on the observations to be standardized (pressure head) is of course open to objection. Nevertheless, the data which Dawson offers are of interest, and may be not far from correct, since his animals were probably studied under comparable conditions and the velocity head in the femoral artery under these conditions probably did not vary greatly in the different animals, with the result that the imperfection of the standard was similar although not constant throughout.

With regard to the difference between femoral and carotid pressures, Dawson has the following footnote (p. 256).

> It is an old observation, made by Hürthle with his elastic manometer, that the systolic end pressure in the femoral is greater than that in the carotid (*Archiv für die gesammte Physiologie*, 1890, xlvii, S. 32–34). An explanation of the phenomenon has been offered (Von Kries, Studien zur Pulslehre, Freiburg, 1892, S. 67–68), but seems not to be entirely satisfactory. At present, however, no new explanation will be attempted.

The higher systolic end pressure in the femoral is to be expected from the fact that it measures pressure head plus velocity head, and that the increment due to the latter may be increased by the effect of water hammer.

Dawson makes the empirical observation (p. 253) that "the end pressures in the axillary and brachial arteries are about equal to the lateral pressures of the aorta at the origin of the subclavian artery." (On page 256 he says:) "The conclusion reached by Erlanger on purely *a priori* grounds, namely, that the pressures determined in the brachial artery by means of his instrument may be regarded as equivalent to the lateral pressures in the aorta at the origin of the subclavian, is almost certainly correct." This conclusion is, of course, in strict keeping with the general principles of hydraulics considered above.

The maximum pressure at various points along the aorta is indicated by the curve marked S in Figure 4. One can readily see that there is a gradual fall of the pressure head as one proceeds distally along the aorta. In a system of rigid pipes with water flowing steadily, the

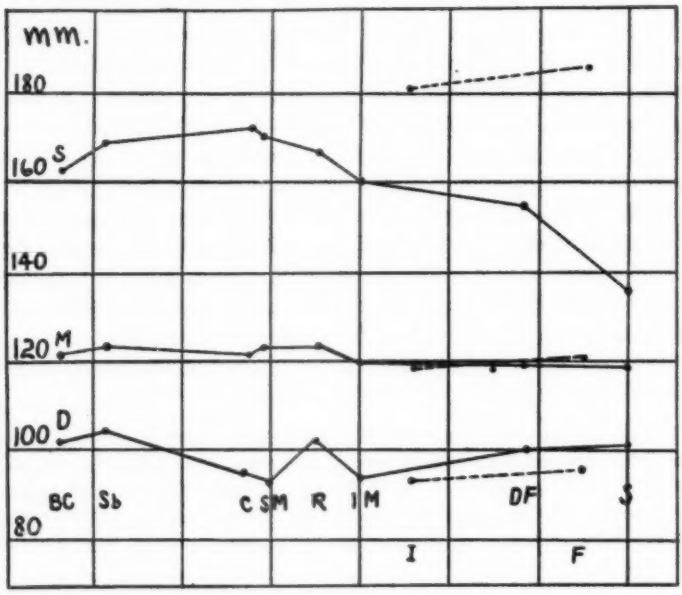

FIG. 4. AORTICO-FEMORAL SYSTEM

S, M, and D are the systolic, mean, and diastolic end pressures, respectively. The solid lines connect end pressures taken in side branches of the aortico-femoral trunk. Broken lines connect pressures taken in the main trunk of the system. BC is the brachio-cephalic artery; Sb, left subclavian; C, coeliac axis; SM, superior mesenteric; R, left renal; IM, inferior mesenteric; I, iliac; DF deep femoral; F, femoral; S, saphenous. (Taken from Dawson.)

pressure head at any given point depends on the resistance to be overcome between that point and the point of exit of the water. The fall in pressure along the aorta is partly explained by the resistance which has already been overcome when the blood has reached the more distal portion. We do not believe, however, that the resistance is the only

factor which accounts for this loss of pressure head, because in so wide and short a pipe as the aorta the resistance is comparatively small. The higher pressure head proximally is partly explained by the inertia of the column of blood and its tendency to resist acceleration. The nearer the blood is to the base of the aorta, the fewer are the avenues of escape through lateral branches, and the more likely is the aorta to be distended. The more distal portion of the aorta is less likely to be distended and its pressure head less likely to be increased because the blood, which during systole is forced into the aorta, has so many more arterial branches through which to escape. So far as the fall of pressure head along the aortico-ilio-femoral line is concerned, this factor is probably more important than the loss of head due to friction.

As one proceeds distally along the aorta there is also a loss of velocity head. If the aorta were a rigid tube of uniform diameter with no branches and with a steady flow of blood, the velocity would have to be the same throughout. When lateral branches are introduced, some of the blood escapes through these, with the result that less blood has to pass through the more distal portion of the aorta and consequently its velocity must be less. The greater dilatation of the aorta near its root due to higher pressure head affords a wider bed for the flow of blood and tends to decrease the velocity at that point. The two factors influence velocity in opposite ways and the final result depends upon the quantitative effect of each. *A priori* it seems likely that the factor of lateral escape is more effective than that of proximal dilatation, and that the velocity head at the lower portion of the aorta is less than the velocity head near the root.

The highest maximal pressures which Dawson records are those for the left iliac, 183 mm. of Hg., and for the left femoral 188 mm. of Hg. The physical explanation for these high values is that these are the only arteries where the full velocity of the stream has been met, head on. The pressure head found at the point of exit of the coeliac (171 mm. Hg.) seems a little high for its position and this may be due to the fact the coeliac artery is not directly perpendicular to the aorta (like the renal) but descends somewhat and receives some of the velocity head along with the pressure head from the aorta.

Another outstanding finding is that the pulse pressures found in the femoral and iliac arteries, (reported as 93 and 91 mm. of Hg., re-

spectively) far exceed the pulse pressures in any of the lateral arteries. The pulse pressure as here determined is the difference between the maximal and minimal pressures. The femoral and iliac arteries, as compared with the lateral arteries, have a much higher maximum and a slightly lower minimum pressure. The higher maximum pressure in these arteries is easily understood on the basis of the high systolic velocity head. The lower minimum pressure is probably due to the greater tendency toward diastolic relaxation in these arteries because of the greater rapidity with which the blood escapes from them during systole.

In general, the value of Dawson's findings is that they illustrate the applicability to the circulation of the laws of hydraulics. These laws may be applied to the flow of water through rigid pipes, and one must constantly bear in mind that the arterial walls are distensible. It is for this reason that we have confined our attention largely to the readings of maximal pressure because when the pressures in the arterial tree are at a maximum the arteries are most resistant to further distention, and then arterial blood flow for the brief space of time closely resembles the flow of water through rigid tubes.

His values illustrate, first, the fall of pressure head as one proceeds toward the periphery, and secondly, the effect of velocity head in the vessels that form a direct continuation from the aorta in the direction of flow. The only objection to the data is that they have been distorted slightly by using the femoral standard.

It is only during the brief portion of systole when the whole arterial system is well distended, and the resistance of the arterial walls to further distention is considerable, that the blood flow resembles the flow of water under pressure through rigid pipes. At the beginning of diastole, when the heart has ceased to discharge its blood into the aorta, and the blood because of its velocity head continues to leave the arterial system, the arterial blood volume rapidly decreases, the distention of the walls is diminished, and the pressure head falls in the whole arterial tree. At any particular moment, since there is a loss of head due to resistance as the blood proceeds to the periphery, the pressure head is higher in the more proximal than in the more distal portion of the aorta. Allowances must be made for changes in the cross-section area of the stream, as well as losses of head due to changes of direction and passage through a constricted orifice.

The maintenance of a pressure head in the arterial system depends not only upon the resistance to be overcome but also upon the presence within the arteries of sufficient blood to fill and somewhat distend them. The ability of the aorta and large arteries to stretch during systole permits the arterial system to hold more blood than the normal resting capacity. During diastole the aorta and arterial walls contract, encouraging the blood flow from the arterial system and maintaining a pressure head. The wider distention of the arterial walls during systole indicates that the intra-arterial pressure head is higher during systole than during diastole.

Since the arterial, arteriolar, and capillary channels which form the peripheral resistance are fairly constant so far as surface friction is concerned, we must explain why it is possible for the intra-arterial pressure head at any point to be higher during systole than during diastole. This is due partly to the fact that resistance increases with increased velocity and above the critical point is proportional to the square of the velocity. The greater the difference between systolic and diastolic velocities, the more significant is the change of resistance. The more important point, however, is the inertia of the blood in the arterial system, and the readiness of the arterial walls to expand during systole to accommodate a goodly portion of the blood which is pumped from the left ventricle. This inertia prevents the arterial blood from taking up the velocity that would be necessary to permit the blood to escape from the arterioles as rapidly as it enters the aorta. The inertia of the arterial column, or tendency to resist acceleration, forms an obstacle to the flow of blood and permits the systolic pressure head to rise above the diastolic at any particular point in the arterial tree.

From Dawson (pp. 251–2).

	MAXIMUM PRESSURE	MINIMUM PRESSURE	PULSE PRESSURE
Left femoral	188	95	93
Left carotid	162	103	59*
Left subclavian	168	105	63
Deep femoral	152	102	50
Saphenous	134	102	32

*It seems that this value is erroneously recorded as 49 in the original paper.

The above figures will give us some idea of the cyclic changes that occur in the arterial system of the anesthetized dog. The left subclavian forms an excellent piezometer for determining changes of pressure head in the aorta. The effect of water hammer in increasing the maximal reading obtained in the left subclavian is probably small enough to be neglected. The readings here indicate that the intra-aortic pressure head falls from a systolic maximum of 168, to a diastolic minimum of 105, the difference ("pulse pressure") being 63 mm. of Hg.

The cyclic changes in pressure head occurring in the femoral artery are indicated by the "pulse pressures" of the deep femoral and saphenous arteries. From examining the maximal values for these two arteries one can see immediately that the deep femoral gives a maximal value which is too high for the pressure head in the main femoral artery (as indicated by the saphenous artery) and the explanation is that the deep femoral does not come off at right angles from the main vessel, is an imperfect piezometer, and its maximal reading is exaggerated because it includes some velocity head derived from the main artery during systole. It is significant that the minimal values are the same for both the deep femoral and the saphenous arteries, suggesting of course that during that part of diastole when the mercury column stands at a minimum height the velocity of flow is so slow that it does not produce any detectable difference, despite the difference of angles which the two branch arteries in question form with the main artery.

Accepting the values obtained from the saphenous artery as more nearly correct, it follows that during diastole the pressure head in the femoral artery drops 32 mm. of Hg. The "pulse pressure" for the femoral is given in the table as 93 mm. of Hg. The difference is explained by the high maximal pressure obtained in the femoral because of the high velocity head, and because of such increment as results from the effect of water hammer.

The minimal pressure recorded for the femoral is 7 mm. of Hg. below the minimal pressures in the deep femoral and saphenous. This may be due to the imperfect method of standardization; or it may be due to the effect of water hammer in determining femoral end pressures. Water hammer is characterized by an abnormally high pressure head immediately the obstruction is met, this being quickly followed by a phase of rarefaction and reduced pressure. It is possible that by the

use of a minimal valve, the mercury column will respond slightly to this phase of reduced pressure. At present, however, the point is not worth dwelling upon.

The rise of 93 mm. of Hg. in the femoral end pressure, from a minimal diastolic of 95 to a maximal systolic of 188, is divisible into two parts—an increase of 32 mm. of Hg. due to rise in pressure head *per se*, and a further increase of 61 mm. of Hg. due to the increment resulting from velocity head, augmented by the effect of water hammer. The precise values here recorded may not be applied to man without further study, first, because they are obtained from anaesthetized dogs, and secondly, because they have been poorly standardized. For qualitative comparisons, however, they are extremely suggestive.

With regard to the dynamics of arterial flow we may summarize as follows. At any particular point in the arterial tree, there is a cyclic change of pressure head, velocity head and total head, being higher during systole and falling during diastole. At any particular instant during the cardiac cycle, there are differences in pressure head and velocity head throughout the arterial tree, being highest near the root of the aorta and lower as one proceeds toward the periphery. In offering values for blood pressure, experimentally determined, we shall have to specify velocity head, pressure head, or total head, at what point in the arterial tree, and at what time during the cardiac cycle.

The importance of the anatomical relationship of the arteries is seen in that the arteries which arise at right angles to the aorta depend for their blood flow upon the pressure head in the aorta, not being directly effected by changes in velocity head *per se*. The blood flow to the head and arms, for example, depends not merely upon the pressure head in the aorta, but also upon how long this intra-aortic pressure head is maintained. Of this we shall say more later.

THE PULSE

Most textbooks of physiology speak of a pulse wave which is defined as a wave of pressure arising at the heart during systole and being propagated toward the periphery (Howell p. 519). By determining the time of its appearance at different points in the arterial tree, and measuring the distance between these points, the velocity of the wave is found to be about 7.5 meters per second. By measuring its duration at any point, the length of the wave is found to be 3.25 to 4.5 meters.

Wiggers (p. 190): As this increase in pressure is transmitted toward the periphery the arterial distention is also communicated from one segment of an artery to the next peripheral segment in the form of a wave, the velocity of which is entirely independent of the blood flow. This is evident from the fact that the pulse wave is transmitted peripherally at a velocity of some 6 to 10 meters per second, while the average velocity of the blood flow is probably 0.3 meter per second. The relation of the pulse wave and the blood flow may be compared to an impact transmitted through a train of cars in motion; the rate at which the impact travels is entirely independent of the speed of the train, and may, in fact, occur in an opposite direction."

Wiggers points out that the central pulse (subclavian) is different in shape from the peripheral (radial), and (p. 199) that the difference is due to the fact that "these complicated series of pressure variation present in the aorta and large arteries are modified in their peripheral transmission by *friction and interference with reflected waves* (Frank)."

To say that the pulse, which really represents the cyclic changes in pressure head at any particular point in the arterial tree, represents a pressure wave, is an erroneous application of the term, and implies the inadequacy of the ideas which are now generally entertained regarding the exact nature of the pulse, the method of its propulsion, and the factors which determine the velocity of its advance, as well as its general form.

Every standard textbook of physics discusses the nature of waves and of wave motion. Kimball (p. 180): "A wave may be defined as a form or configuration of motion advancing with a finite velocity through a medium.

"By means of waves energy is transmitted, being passed along from one part of a medium to the next by the interaction of adjoining parts."

A pressure wave may be made to pass along a "non-compressible" fluid, which completely fills a "non-distensible" pipe, by suddenly increasing the pressure at one end of the pipe. This tends to reduce the volume and increase the pressure of the fluid at the end where the pressure is applied, and the affected fluid is now no longer in equilibrium with uncompressed fluid adjacent to it. The compressed fluid, therefore, expands and compresses the adjacent fluid, which in turn

expands and passes on the state of compression to the more distant adjoining uncompressed fluid. A state of compression, therefore, starts in the fluid at one end of the pipe and is conveyed along the column of fluid in the form of a pressure wave. The wave of increased pressure is followed by a wave of decreased pressure. The fluid particles at any point continue in cyclic harmonic motion, similar to the swing of a pendulum, but they do not advance along the pipe and when the wave dies out, the particles remain exactly where they were at the outset. The initial expenditure of energy to start the wave has been "lost" because of friction, and if there were no friction involved, the wave would continue indefinitely.

When the wall of the tube is distensible to any degree, even the slightest increase of pressure at any point (say the radial artery) will tend to distend the artery. Since the compressibility and expansibility of the blood are negligible compared with the distensibility of the artery, the actual distension of the artery presupposes two things, first, an increase in intra-arterial pressure, secondly, a considerable increase in the amount of blood locally to fill and distend the artery. Assuming even that a wave of pressure could be made to pass along the arterial blood, such a wave could never distend the artery sufficiently to make the excursion felt, without an increase in the amount of blood sufficient to expand the artery from its diastolic to its systolic volume.

We reach the inevitable conclusion, therefore, that in a system of distensible arterial tubes, the advancing line of increased pressure must be accompanied by the advancing line of increased blood volume locally, and that the former cannot precede, but must accompany, the latter. The radial pulse, therefore, depends not merely upon propagation of pressure but also upon actual mass movement of blood. A *true pressure wave* travels along a medium independent of mass movement and may be opposite in direction. Can the reader conceive of *a pulse wave* traveling centrally along the radial artery from the hand towards the elbow, while the blood is flowing in the opposite direction?

Wiggers (p. 198) shows the difference in form between subclavian (central) pulse and radial (peripheral) pulse in man. If they both represent the same pressure wave, how can they differ so widely in configuration, in duration, hence in velocity, or in length? These differences are explained by Frank as being due to modification during

"peripheral transmission by *friction* and *interference with reflected waves*." (Quotation and italics from Wiggers.)

The present writer believes that pressure waves play little or no rôle in the normal arterial circulation and that the pulse in the arterial system (say at the radial) is an expression of local changes in pressure, blood volume, and arterial diameter, during the cardiac cycle. The nature of the pulse, its origin, and its form of propagation mark it off as entirely different from a pressure wave, and any attempt to apply formulae regarding wave length, velocity, and duration, to a study of the pulse is patently an error based on a misconception.

During the period of ejection, blood is being discharged from the ventricle into the aorta. Synchronous with the discharge, and progressing with it, is an increase in the intra-aortic pressure, an increase in its diameter and in the amount of blood it contains. As the systolic discharge from the ventricle continues, volume increase, pressure increase, and forward-movement of blood continue, and the length of the arterial system (measured from the root of the aorta), showing these changes, increases during the ejection period. Systolic discharge continues even after the pulse has reached the radial artery.

On being ejected into the aorta, the blood will either distend the aorta or push forward the blood in front of it. The facility of the former depends on the distensibility of the aortic wall, which steadily decreases with continued distention. The difficulty of the latter depends upon the inertia and resistance which oppose the forward movement of the arterial column of blood.

During diastole the aortic walls are comparatively lax, and at the beginning of the ejection period much of the blood goes toward distending the aorta. This is quickly done, however, because the velocity of ejection is much greater at the earlier than at the later part of the ejection period. During the later part of the ejection period, with the aorta considerably distended, the ejected blood to enter the aorta must cause relatively more forward movement of the arterial column than it did during the early part of the ejection period. This the heart is well adapted to do because the ventricular wall can develop a higher pressure head during the later part of the period of ejection than it can during the earlier.

The changes which occur in the aortic tree, therefore, during the

ejection period, are, first, a proximal dilatation and pressure increase with but slight forward movement of blood. Very quickly, however, the forward movement of blood becomes prominent and, advancing toward the periphery, is the distal end of the proximal portion of the arterial tree in which the pressure and volume have been increased and the wall distended. This advancing distention when felt over the radial or dorsalis pedis is perceived as the pulse. 'Tis true, it advances from heart to periphery, and its average velocity may be measured for a given portion of its course, but its resemblance to a pressure wave is really of the most superficial variety.

HAEMODYNAMICS OF AORTIC INSUFFICIENCY

In a well developed case of aortic insufficiency which has been present for some time, perhaps the most striking anatomical change is the extraordinary hypertrophy of the left ventricle. The mechanism of contraction and the forces concerned are precisely those found in the normal heart, except that the cardiac contractions are more forcible and the cyclic changes are somewhat exaggerated.

The work of contraction of this powerful pump, as in the normal heart, is spent in overcoming peripheral resistance and imparting velocity to the ejected blood. Since during a single contraction the hypertrophied left ventricle does more work than the normal ventricle, and since we have no reason to believe that the peripheral resistance is materially increased in aortic insufficiency, it follows that the extra energy of contraction of the hypertrophied ventricle is spent in imparting increased velocity to the ejected blood. The factors in aortic insufficiency making for velocity of the stream are the hypertrophied ventricular musculature, the increased systolic output, and possibly the decreased ejection time. The most important factor, however, is the low intra-aortic diastolic pressure.

Consider a small cube of blood in the position A (fig. 5) at the very beginning of ventricular contraction when the intra-ventricular pressure has risen to the diastolic aortic pressure, say 20 mm. of Hg. At that instant the cube remains motionless, because the forces acting upon it are in equilibrium. The intra-ventricular pressure rapidly increases and the force exerted accelerates the cube in the direction of the arrow. This accelerating force at any particular instant depends

upon the difference of pressure in front of and behind the cube, and in aortic insufficiency is obviously much greater than normal because of the low diastolic intra-aortic pressure, and secondly because of the increased systolic intra-ventricular pressure, the latter being due to the dilatation and hypertrophy of the left heart (Starling's law of the heart).

Hewlet and Van Zwalewenburg (6) have demonstrated by use of a plethysmograph that in cases of aortic insufficiency the velocity of flow in the brachial artery reaches abnormally high levels. From

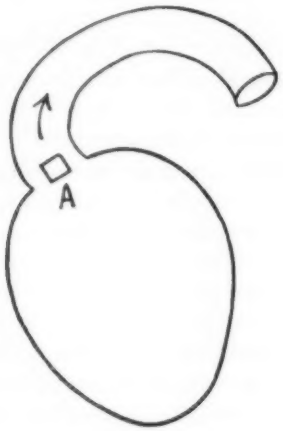

FIG. 5

our studies of arterial haemodynamics we should expect the increased velocity to be demonstrated even more strikingly in the femoral and popliteal arteries, since these arteries are direct continuations of the aorta.

In the normal circulation the velocity of the arterial blood undergoes some cyclic variation, being higher during systole, but the stream is fairly steady and the arterial system is full and somewhat distended all the time. The pressure head, too, shows some cyclic variation, being higher during systole, as indicated by the sphygmogram, but here too the variations probably are not extreme. Indeed, if we assume that the maximal systolic pressure in the femoral artery of a

normal man is 120 mm. of Hg. that the minimal diastolic pressure is 80 mm. of Hg. at the same point, it follows that the total head has dropped from 120 mm. of Hg. systolic to 80 mm. of Hg. diastolic. We have reason to suppose that during diastole both the velocity head and the pressure head are reduced, and we may therefore conclude that the diastolic loss of pressure head or of velocity head must each be less than 40 mm. of Hg. because their sum is equal to that amount. In the normal circulation loss of head is due in large measure to loss of pressure head, as indicated by the post-dicrotic fall of the catacrotic limb in the sphygmogram.

In aortic insufficiency these factors are markedly altered and the alteration consists merely in this—that the normal cyclic changes in pressure head, velocity head and total head of the arterial blood are strikingly exaggerated. The reason for this becomes apparent when we consider the anatomical and physiological pathogenesis of the lesion. Immediately the aortic cusps are rendered incompetent, a fairly large amount of blood regurgitates into the left ventricle during the diastole which follows the introduction of the lesion. The left ventricle then finds itself filled with more then the usual amount of blood because the auricle has made its normal or nearly normal contribution. The left ventricle, being abnormally distended, contracts more forcibly during the following systole in its effort to empty itself; this has been well demonstrated by Starling and illustrates the so-called law of the heart. As long as the heart muscle is normal it will obey the "all or none" law of contraction usually attributed to cardiac muscle, and as long as the heart can handle its load, the actual force of contraction will depend upon the degree of distention during diastole, *i.e.*, upon the amount of blood which the ventricle must eject. The force of ventricular contraction in aortic insufficiency is further increased, as Stewart has suggested, by the increased intra-ventricular diastolic pressure resulting from aortic incompetence and the tendency of the intra-ventricular diastolic pressure to become equal to the diastolic pressure within the aorta, with which the ventricle is always in communication. The correctness of these observations is indicated by the extraordinary hypertrophy and dilatation which the left ventricle undergoes when the lesion lasts for any length of time. The results of these changes are apparent. In a well developed case

of aortic insufficiency, the left ventricle during systole ejects more blood than normal, ejects it with more force, and imparts to it a slightly greater pressure head than normal, a considerably greater velocity head than normal, and a total head which exceeds the normal by the sum of the two previous increments. These are the conditions which obtain at the height of systole when the total head is at a maximum.

In this disease, however, not only are the systolic increments exaggerated above normal but also the diastolic decrements are exaggerated below normal. The diastolic pressure head at any point in the arterial system drops far below normal, as indicated by the collapse of the arterial walls during diastole and by the low "diastolic" pressure. The maximal and minimal pressures in the left brachial artery may be taken to indicate the maximal systolic pressure head in the aorta at the point of exit of the left subclavian and the minimal diastolic pressure head at that point. While the normal values are usually about 120/80, the values in aortic insufficiency may be about 140/30. The systolic pressure indicates the maximal systolic pressure head in the aorta. The diastolic pressure is very likely merely an approximate index of the lowest diastolic pressure head in the aorta, because, as will be seen, it does not measure the minimal diastolic total head in the brachial artery. Disregarding these refinements, however, we are correct in assuming that the cyclic changes of pressure head in a case of aortic insufficiency are of extreme degree, and that the collapsing pulse is one of its manifestations. The reason for the very low intra-arterial diastolic pressure head is that the systolic velocity is great enough to permit rapid emptying of the arterial system and the arteries tend to collapse because there is insufficient blood to distend them. During diastole, the arterial emptying is accelerated by aortic regurgitation.

The cyclic changes in velocity head are also extreme in aortic insufficiency. That the systolic velocity head is increased may be suspected from the pathological physiology as discussed above, may be accepted as probable because of the high systolic pressure in the popliteals, and because of its ability to explain so many of the circulatory changes associated with the lesion.

The diastolic velocity head is remarkably reduced, and may fall to zero or below in some parts of the arterial system. Since the normal

diastolic flow or velocity head is maintained by the contraction of the distended aorta and arterial walls, and since in aortic insufficiency during the latter part of diastole at least the arterial system is abnormally "empty" or considerably less distended than normal, the usual mechanism for the maintenance of velocity head is absent. In such parts of the arterial system, *i.e.*, near the root and arch of the aorta, where there is most likely to be diastolic regurgitation, the velocity head drops down to zero and below, because the direction of flow is reversed. In any part of the arterial system whether or not there is a diastolic reversal of flow, we are correct in assuming that the forward velocity is less during the latter part of diastole at least than it is in the normal circulation during the corresponding period.

The cyclic variations of intra-arterial total head are easily deduced from the above considerations regarding pressure and velocity head, for at any particular moment the total head will be equal to the sum of pressure and velocity head.

The above considerations give us some idea of the differences in pressure head and in velocity head at different points in the circulation. They also emphasize the cyclic changes in both pressure head and velocity head at any given point during cardiac systole and diastole. The term 'blood pressure' is ambiguous unless we specify velocity head, pressure head, or total head—at a given point in the arterial system and at a given time in the cardiac cycle. Some of these values undoubtedly will prove more difficult to measure directly than to calculate from indirect observations.

Let us pause here for one or two clinical correlations. We have observed above that, because of the anatomical relationships of the large arteries to the aorta, the blood supply to the head and upper extremities depends upon the pressure head in the aorta, and the amount of blood which the head and upper extremity receive depends also upon *how long* this intra-aortic pressure head is maintained. We have found that the intra-aortic pressure head undergoes extreme cyclic variations in this disease and that, although the maximal systolic pressure head may be increased only 20 mm. of Hg., the minimal diastolic pressure head may be reduced 50 mm. of Hg. and that the high pressure head is only momentarily maintained, with the result that the average intra-aortic pressure head during both systole

and diastole is greatly reduced. From this we may conclude that in aortic insufficiency the blood supply to the head is considerably reduced, despite the increased velocity, largely, indeed, because of the increased velocity during systole. This *a priori* conclusion that the blood supply to the head is reduced is borne out by the facial pallor of these patients, by the frequency of mental symptoms, and possibly by their extraordinary dyspnoea when the velocity flow is increased by exercise of any sort.

Another observation which may be of clinical importance is the extreme intra-arterial cyclic changes of pressure head. That with each systole of the heart the distending force acting upon the arteries should rise from its abnormally low diastolic level to its abnormally high systolic level indicates to what extraordinary strain the arterial walls are subjected. Is this why so many patients with aortic insufficiency develop arteriosclerosis? Does a high pulse pressure, even in the absence of aortic insufficiency predispose to the development of arteriosclerosis? A collapsing pulse seems to cause arterial trauma as well as cerebral anemia.

Let us now consider briefly the questions of collapsing pulse and of regurgitation. H. A. Stewart (7), in an excellent experimental study of aortic insufficiency in dogs, reached certain conclusions which are of sufficient importance to bear repeating. First, he confirmed Henderson's finding that in the normal heart the ventricle becomes filled early in diastole, almost as rapidly as it empties itself. The ventricle then remains quiescent until the onset of the following systole of the auricle which "ejects only a fraction of a cubic centimeter of blood into the already filled ventricle." He also demonstrated that the rapid fall of pressure, indicated by the rapid fall of the catacrotic limb in the sphygmogram, takes place for the most part during systole, since it occurs before the dicrotic notch. Hence, the fall in pressure occurs before the diastolic regurgitation and cannot be caused by it. He concluded therefore that the collapsing pulse is due not to regurgitation but rather to an increased blood flow through the capillaries. To explain the increased flow, Stewart accepts Starling's suggestion that there is a reflex vaso-motor dilatation to save the heart from over-exertion. Against this hypothesis of a vaso-motor dilatation to explain the rapid egress of blood from the

arteries, we may say, first, that there is no convincing evidence that it occurs; secondly, that it is not in keeping with the striking pallor found in these patients; furthermore, a vaso-motor dilation would markedly decrease peripheral resistance, and since the vessels leaving the arch of the aorta may be used as piezometer tubes, they ought to show a fall in systolic pressure early in the disease, but it is found, experimentally and clinically, that in aortic insufficiency the systolic pressure in the brachials remains the same or is slightly increased. When arteriosclerosis sets in, the pressure in the arms may be considerably increased. It is apparent that a vaso-motor dilatation of even brief duration would greatly decrease the blood supply to the brain, and this may explain the attacks of dizziness and faintness and other cerebral symptoms from which these patients so often suffer. Such transitory periods of cerebral anemia possibly explain the attacks of paroxysmal dyspnoea, so often nocturnal, compelling the patient to leave his bed and go to the open window to "fight for air."

We agree with Stewart, therefore, that the systolic fall in pressure (which by the way is simply intra-arterial pressure head, as measured by the distention of the vessel wall while the blood continues to flow, the velocity head being undisturbed) as indicated by the abrupt descent of the catacrotic limb before the dicrotic notch is reached, is the result of the increased flow of blood through the peripheral vessels. Such increase, however, is not due to reflex peripheral vaso-dilatation but rather to the increased velocity head of the blood imparted to it by ventricular contraction, the peripheral resistance in uncomplicated cases remaining practically unaltered.

The capillary pulse, so-called, is also a manifestation of the velocity with which the blood is ejected into the smaller vessels and capillaries as a result of systolic contraction. As we watch the nail-bed in a patient with aortic insufficiency we see a sudden systolic flushing of the pale areas, which means that the blood vessels, which give the reddish hue to the nail-bed in these areas, during diastole are comparatively empty and the area appears pale. With the sudden systolic ejection of blood, however, they become filled and there is a transitory reddening of the area because the rapidity of ejection lasts only for an instant, and the ejected blood passes on into the larger collecting veins which lie deeper. Arterial flow to the head and arms in this

disease occurs in sudden spurts rather than as a steady flow, because the intra-aortic pressure head collapses so early in the cardiac cycle, and reaches so low a level.

The question of regurgitation in aortic insufficiency has during the past two decades been a subject of disagreement among physiologists and clinical cardiologists. H. A. Stewart has given us a short history of the problem, and C. Wiggers (8) reviewed the literature up to 1915 and offered his own experimental findings with interpretations. Without making any pretense at completeness, we shall very briefly summarize the question, and offer such interpretations as seem to us reasonable.

Since the publication of H. A. Stewart's paper in 1908 many have insisted upon the distinction between aortic insufficiency and aortic regurgitation. On the basis of his experimental studies of aortic insufficiency in dogs, H A. Stewart concludes that the volume of blood which regurgitates is negligible. The tendency toward regurgitation, he points out, is reduced because the ventricle early in diastole is rapidly filled with blood from the auricle. Furthermore, the blood pressure in the aorta at the end of systole is usually not more than 15 mm. of Hg. higher than the intra-aortic pressure at the end of diastole. "The difference in pressure between the systolic end pressure and that at the end of diastole is accounted for partly by the loss due to regurgitation and partly by capillary escape. Under no circumstances, then, can this fall of pressure due to regurgitation exceed or even equal this difference." This reasoning to us seems quite correct. The author, however, goes on to state that after the production of the lesion, the systolic output is increased by only 0.5 cc. "There was no diminution in the amount of blood delivered by the auricle, as was evidenced by the fact that there was no dilatation of its cavity.

"The facts admit of but one interpretation, *viz.*, that after an extensive valvular lesion the actual amount of blood which regurgitates is fractional in quantity as compared with that which enters naturally from the auricle."

Three years later, W. G. MacCallum (9) published his conclusions after studying the results of experimental aortic insufficiency in dogs. "The tearing of an aortic valve producing an aortic insufficiency causes the heart to perform an extraordinary amount of work, part of which is

devoted to the maintenance of nearly a normal rate of flow in the vessels, while the remainder is expended upon the forcing out of a quantity of blood which each time regurgitates through the torn valve. From all this, it seems unavoidable to conclude that regurgitation plays a prominent part in the whole complex of aortic insufficiency."

Wiggers, on the contrary, states that "all experimental evidence contradicts the view so prevalently held by clinicans that a large portion of the systolic discharge flows back in each diastole.

"The essential dynamic disturbance, *viz.;* the collapsing pulse, is brought about, not by the volume of blood which regurgitates, but by the *regurgitation of pressure* during diastole."

Wiggers (p. 554 (5)): "The essential dynamic disturbance, *viz.*, the collapsing pulse, is brought about, not by the volume of blood which regurgitates, but by the regurgitation of pressure during diastole. While the relatively small orifice in the insufficient valves, together with the rapid natural filling of the ventricle, prevents any considerable regurgitation of blood, it permits an equalization of pressure. This conception of the regurgitation of pressure without any appreciable back-flow of fluid may be clarified if we suppose a system consisting of two flasks, A and B, communicating by a rigid tubing and a stop-cock, to be completely filled with fluid. Let us suppose, further, that the stop-cock is closed and that the fluid in flask A is placed under a pressure of 5 mm. of Hg. and that in flask B under the pressure of 150 mm. of Hg. Upon opening the stop-cock, the pressure in the two flasks will become equal, that in flask A rising, that in flask B falling. It is obvious that since both flasks are already filled with an incompressible liquid, no transfer of liquid can take place. If both flasks were partially distensible, then a small transfer of fluid could take place."

Let us first examine the evidence upon which H. A. Stewart bases his conclusion that "the actual amount of blood which regurgitates is fractional in quantity." The facts which Stewart gives us are that the systolic output is about 12 cc. *before* the production of the lesion, 12.5 cc. *after*. There is no evidence of cardiac failure either before or after the aortic valve has been rendered insufficient. We should be compelled to admit that the regurgitation must be equal to 0.5 cc. if the Henderson ventricular plethysmograph which Stewart uses for deter-

mining systolic output surrounded *only the left ventricle*. On the contrary, it surrounds both ventricles, and the difference merely means that both ventricles together eject 0.5 cc. more blood after the lesion than before the lesion is introduced. With competent aortic valves there is no regurgitation and each ventricle must eject the same amount, *i.e.* 6 cc. After the valve has been injured, it is possible that the right ventricle ejects 6.0 cc. and the left 6.5. cc., with 0.5 cc. regurgitating. The facts, however, admit of another interpretation. It is possible that the right ventricle ejects 5.0 cc. and the left 7.5 cc., with 2.5 cc. regurgitating. It can readily be seen that the net output of the left ventricle is still equal to that of the right and that the total systolic output is still 12.5 cc. and agrees with Stewart's figures, although the amount of blood regurgitating far exceeds his estimate. Indeed, Stewart points out from his volume curves (p. 116) that after the lesion has been introduced the ventricle fills somewhat faster and that the acceleration involves only the commencement of diastole proper. This, as we see it, simply means that, early in diastole, the ventricle is being filled from both the auricle and the aorta, and we have reason to suppose that the auricle contributes much less than normally and that the aorta contributes more than Stewart believed. These considerations explain why W. G. MacCallum, who actually measured the systolic output as the blood escaped, should have calculated that the regurgitation was considerable.

With regard to Wiggers' analogy of the flasks to explain a regurgitation of blood, we may say that in the first place the analogy attempts to explain a conclusion which to our mind does not follow the given premises. Furthermore, the analogy is objectionable for the following reason. When one connects two rigid flasks with a rubber tube, it is possible to equalize a marked disparity of pressure in these flasks by allowing the transfer of a small amount of fluid from the flask of higher to the flask of lower pressure. The actual mass of fluid passing from one flask to the other depends upon its compressibility under pressure. If the fluid be a gas, a considerable amount will have to change position. If the fluid be water or blood only a very small amount will have to flow through the open stop-cock. When, however, one is considering an elastic musculo-membranous container such as the aorta, conditions are quite different. The pressure head depends upon

how much the aorta is distended, and the pressure head can be reduced only by providing an avenue of escape for the blood which is distending it. The blood escapes, of course, distally along the aorta, through the lateral branches, and by regurgitation in diastole. The objection to the analogy is, therefore, that one can cause very great pressure changes in a rigid flask by forcing in or drawing out very small amounts of a poorly compressible fluid. In an elastic musculo-membranous distensible container one must introduce or withdraw considerable amounts of a poorly compressible fluid to produce comparatively small changes of pressure.

DYNAMICS OF REGURGITATION

The blood which regurgitates into the left ventricle during diastole is the blood which is near the base of the aorta and which has been the last portion of the systolic output. The force urging the blood back into the ventricle is the excess of intra-aortic pressure head over intra-ventricular pressure head. The "backward velocity" equals the backward accelerating force multiplied by the time during which that force acts. The backward velocity will be able to cause the blood to regurgitate only after its magnitude is such as to overcome the forward velocity possessed by the blood at the end of systole.

There are several factors which might reduce the amount of regurgitation. First, a high forward velocity head at the end of systole; secondly, a low intra-aortic pressure head at the beginning of diastole; thirdly, a rapid rise of intra-ventricular pressure head in early diastole; fourthly, a short diastolic time.

It is of peculiar interest that the anatomical and functional changes which the circulatory system undergoes in response to an incompetence of the aortic valves are precisely those which were mentioned above and which readily suggest themselves according to the simple laws of kinetics.

From our previous considerations of cardiodynamics it seems reasonable to suppose that the velocity head of the ejected blood is lower at the end than at the beginning of the ejection period; but it also seems probable that the velocity head at the end of systole is higher in aortic insufficiency than during the corresponding period in the normal person. This may be assumed because of the greater force of ventricular con-

traction and the high intra-ventricular pressure head toward the end of systole. Furthermore, the end-systolic intra-aortic pressure head is much lower in aortic insufficiency than in the normal person, because the arterial pressure head begins to collapse early in systole, the reason being the rapid egress of blood from the arteries and the early absence of sufficient blood to distend them.

The third factor, *i.e.*, the rapid rise of intra-ventricular pressure during the early part of diastole has been shown by Henderson and Stewart to be encouraged by rapid inflow from the auricles.

Stewart has pointed out the increase in diastolic ventricular tone and the high diastolic intra-ventricular pressure in aortic insufficiency. This, of course, is to be expected because the aortic lesion keeps the ventricular cavity in communication with the aortic cavity during diastole, and as soon as the left ventricle relaxes at the end of systole, blood will flow in first from the auricle and very quickly from the aorta too, and the pressure in the ventricle will rise to the pressure in the aorta. The left ventricle, therefore, is kept under considerable tension and tone during diastole, and the post-systolic period of relaxation is much shorter and much less complete than in the normal circulation, all this being despite the increase in size of the left ventricular cavity.

Since the force which causes regurgitation during diastole depends upon the excess of intra-aortic pressure head over intra-ventricular pressure head, the decrease in the former and the increase in the latter both reduce the size of the regurgitating force and tend to diminish the amount of blood which regurgitates.

The duration of diastole may or may not be of importance, depending upon the dynamics of the individual case. Ordinarily, the regurgitant murmur is limited to early part of diastole. If we assume that the cessation of the murmur indicates that regurgitation has stopped we must conclude that the intra-ventricular pressure head has become equal to the intra-aortic pressure head, and the force making for regurgitation is no longer present. Under these circumstances, an extension of the diastolic time has no effect upon the amount of regurgitation, and may conceivably permit an end diastolic escape of blood from the ventricle as the intra-aortic pressure head continues to fall because of peripheral escape.

In such cases, however, in which the regurgitant murmur continues throughout diastole, the amount of regurgitation will be reduced by shortening the diastolic period. The end diastolic regurgitating blood will thus be turned about in its course by the advancing blood of the rapidly succeeding systolic contraction.

These, then, are the factors in the circulatory adjustment which tend to reduce the amount of regurgitation. To what extent they succeed during life it is difficult to say, but their effectiveness depends upon the size of the lesion, and the ability of the heart to hypertrophy. At autopsy the amount of regurgitation prior to death is roughly indicated by the difference in size of the right and left ventricular cavities. When there is no regurgitation, the average systolic output of the two ventricles must be equal, otherwise there would be either an accumulation of blood in the pulmonary circulation when the output of the right ventricle exceeded that of the left, or an accumulation of blood, in the systemic circulation when the output of the left ventricle exceeded that of the right. Assuming that there are no abnormal pulmono-systemic communications and that the great hypertrophy and dilatation of the left ventricle indicate that the average systolic output is greater on the left than on the right side, it follows that, as long as the blood in the systemic circulation is not increased at the expense of the blood in the pulmonary circulation, the average amount of diastolic regurgitation must be equal to the difference in average systolic output of the two ventricles.

PRESSURE IN THE LEG IN AORTIC INSUFFICIENCY

The observation which stimulated the writer to begin his studies on haemodynamics was the finding that the systolic blood pressure in the legs of a medical patient with aortic insufficiency was about 100 mm. of Hg. higher than the systolic pressure in the arms. Recalling current notions regarding a fall of pressure as one proceeded to the periphery, we considered it paradoxical that the pressure in the legs (being at a greater distance from the heart) should be so much higher than that in the arms, the influence of gravity being avoided of course by keeping the patient in the horizontal position during the measurement of blood pressure.

Reference to the literature revealed that the high popliteal blood

pressure in aortic insufficiency of some duration is so striking and so constant as to be considered pathognomonic.

From Hill and Rowlands (10) (p. 222). "It has been found by Hürthle and others that the systolic pressure in animals may be higher in the femoral than in the carotid artery.

"Tigerstedt sought to explain this by supposing that the primary pulse wave is reflected from the periphery and adds itself to the primary without change of sign. This, we believe, is due to better conductance of the crest of the systolic wave to the femoral artery. In the healthy young men, the conductance seems to be about the same. In aortic regurgitation the leg arteries are held, we believe, in a contracted state in order that the brain may receive a sufficient supply of blood."

We have already discussed the dynamics which explain the experimental finding that in normal dogs the maximal pressure in the femoral artery is higher than the maximal pressure in the carotid or axillary. In aortic insufficiency this disparity is exaggerated because of the abnormally high maximal velocity which the arterial blood in the aorta attains, although, of course, the *average rate of flow* in aortic insufficiency may be either greater than or less than normal. So far as the head, arms, and kidneys are concerned, the average rate of flow is undoubtedly less than normal, and it will be less than normal not merely in aortic insufficiency but wherever the systolic velocity is high enough to make the intra-aortic pulse (rhythmic change of pressure head) collapsing.

When the pulse is of collapsing type the legs are likely to receive a disproportionately large share of the total output of the heart.

Our attention has been called to H. C. Bazett's (11) recent study of differential blood pressure. Bazett's conclusion is as follows: "These experiments leave little doubt that in the schema the main factor involved is the transformation of kinetic energy into stress, and the unequal distribution through reflected waves of the pressures generated. In general, these pressures will tend to be greater the larger the mass of fluid moving toward a branch. Owing to the closely parallel changes in the schema and experimental animals it is probable that this is also an important factor in the animals, since the mass of fluid of the aorta and common iliacs directed toward the femoral should give a greater kinetic energy than would be present in vessels

of the upper limbs. No adequate explanation has been brought forward for the occasional reversed differential pressure."

We are able to agree only in part with Bazett's explanation. The finding that the differential pressure can be reversed even in a single instance proves that the mass of blood is not the sole or most important factor, for the mass of fluid of the aorta and common iliacs is always greater than the mass of blood present in the vessels of the upper limbs.

We also consider it unnecessary to postulate an unequal distribution of reflected waves, or even their existence, except in so far as they act in the localized mechanism of water hammer.

The differential blood pressure, as we understand it, depends upon the fact that the arteries to the arms and head arise at right angles from the arch of the aorta, that they act as piezometer tubes, and their blood flow depends upon intra-aortic pressure head, variations in intra-aortic velocity head alone not affecting the brachial or carotid blood flow. The femoral pressure, of course, is increased by intra-aortic velocity head because a femoral obstruction meets the aortic stream head on.

As we have found above, there is a fall of pressure head as one proceeds toward the periphery, *i.e.*, the femoral pressure head is exceeded by the intra-aortic pressure head as measured by the brachial pressure (total head). When an animal is in shock, the velocity of arterial flow may be so small that the velocity factor, which ordinarily exceeds the difference between intra-aortic pressure head and femoral pressure head, and which ordinarily gives a higher reading for total head of the femoral as compared with total head of the brachial, will be greatly reduced, whereupon the occasional reversed differential pressure will occur.

SUMMARY AND CONCLUSIONS

The hydrodynamic equation for the flow of water through pipes is briefly discussed and Bernouilli's Theorem is stated.

An application of these principles is made in the attempt to analyze certain problems in haemodynamics.

The forces concerned in cardiac contraction and the influence of respiratory movement are discussed. An explanation for the inspiratory fall of systolic pressure is offered.

Present methods of calculating the work of the heart have underestimated the kinetic energy factor by failing to consider the systolic increase of velocity head imparted to the *whole mass of arterial blood.*

The general applicability of the principles of hydraulics to the problems of arterial flow is demonstrated by their ability to explain and correlate the findings of Dawson on the *Lateral Blood Pressures at Different Points of the Arterial Tree.* Brachial blood pressure is found to depend upon intra-aortic pressure head. Femoral blood pressure depends upon the sum of intra-aortic pressure head and velocity head. Hence the so-called differential blood pressure.

The formation and propagation of the pulse are discussed. The present writer believes that the concepts of pressure waves has been applied erroneously in many problems of haemodynamics, *e.g.*, the nature of the pulse, the excess of femoral over brachial blood pressure, the difference between central and peripheral pulse, etc.

A well developed case of aortic insufficiency is characterized by an exaggeration of the normal cyclic changes in pressure head, velocity head, and total head of arterial blood. Evidence is offered for believing that the maximum velocity head especially increases above normal.

A collapsing pulse diminishes the blood supply to the head, and causes excessive mechanical strain of the arterial walls.

The present writer believes that the collapsing pulse is due to rapid egress of blood from the arterial system, resulting from the abnormally high systolic velocity head.

The conclusion of H. A. Stewart that regurgitation is minimal seems to arise from a misinterpretation of his experimental findings. We agree with W. G. MacCallum that in aortic insufficiency regurgitation plays a considerable rôle. An analysis is offered of the dynamics of regurgitation and the compensatory factors which tend to reduce the amount of blood regurgitating.

The capillary pulse is believed to be a further consequence of the momentarily increased systolic velocity head, and the exaggerated spurt-like character of arterial flow.

The high femoral blood pressure in aortic insufficiency depends upon the high maximum velocity head of aortic and femoral blood. The brachial pressure may remain quite normal because the brachial and subclavian arteries arise at right angles from the arch of the aorta, and

act as piezometer tubes, their flowing depending entirely upon pressure head and not being directly influenced by changes of intra-aortic velocity head.

The difference in mass of blood is only of secondary importance and may exaggerate the effect of water hammer. We consider it unnecessary to postulate an unequal distribution of pressure waves reflected from the periphery, or even their existence, in attempting to explain the excess of femoral over brachial blood pressure.

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CONCERNING THE MECHANISM OF PRODUCTION OF THE KOROTKOFF SOUNDS AND THEIR SIGNIFICANCE IN BLOOD PRESSURE DETERMINATIONS

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The ausculatory method of determining blood pressure was introduced by Korotkoff in 1905. During the decade following the publication of his paper, intensive studies were carried out in various laboratories of physiology to determine the mechanism of production of the compression sounds. As yet, no unanimity of opinion has been reached. Clinicians, however, have been quick to apply a rapid and easy method of blood pressure determination in man, and more or less arbitrarily have adopted certain auscultatory criteria, of which the exact physical significance remains yet to be determined.

The Korotkoff sounds may be heard in the antecubital fossa during decompression of the cuff which has been applied above the elbow.

The changes which these sounds undergo during decompression are quite familiar and may be described in five phases. Wiggers, p. 351 (1):

First phase: The sudden appearance of a clear sound, lasting for a fall of approximately 14 mm. of mercury. This has already been described as a criterion of systolic pressure.

Second phase: The acquisition of a murmurish character, lasting while the pressure falls approximately 20 mm. of mercury more. It is attributed to stenosis of the vessel produced under the cuff at this stage.

Third phase: The replacement of the murmur by a sound becoming progressively louder and lasting during the next 25 mm. of pressure fall.

Fourth phase: The muffling of the sounds lasting while the pressure falls 5 to 6 mm. more. The cause of this muffling has been variously interpreted as due: (a) To the absence of diastolic collapse; (b) to the slowing of the stream under the cuff; (c) to the decreased resonance in the deflated cuff; (d) to a lack of flattening of the arterial wall (MacWilliam and Melvin).

Fifth phase: The disappearance of all sound.

Erlanger (2) presents the following summary of previous theories as well as his own water-hammer hypothesis for the production of these sounds.

"The views held with regard to the origin of the compression sounds of Korotkoff, now extensively employed in the estimation of the arterial blood pressures in man, may for the sake of convenience be divided into two groups:

"(a) Views locating the production of sound in an empty artery beyond the compression. Korotkoff is apparently of the view that compression sounds are due to 'the forcing apart of the wall (of the artery) by the first stream of blood which reaches the artery below the cuff.' He maintains that 'the lower part of the brachial artery, during the time the compression is exerted above it, is in a condition of complete relaxation and that the first blood stream causes a sudden sharp stretching of the wall with the consequent production of sounds.' Korotkoff offers as proof of this contention the fact that a sound is produced when salt solution is poured into the iliac artery of an animal (3). This view or slight modifications of it (4) seem to be held by the majority of those who have devoted some thought to the subject.

"b. Views locating the seat of sound production in the part of the artery compressed apparently are to be found only in the very recent literature. In 1914 MacWilliam and Melvin (5) were forced to this conclusion by the observation that 'the sounds are perfectly well developed and characteristic, when the artery consists merely of a tube in a compression chamber. It might be added here that the changes in form they describe are not those that occur under a pulse of the configuration of the arterial pulse (1). Flack, Hill, and McQueen (6) maintain, without however offering any experimental evidence for their view, that the compressing armlet 'converts the compressed area (of the arm) into a resonating mass, the pulse is not damped down in the labile arteries, but strikes the blood which fills to distention, not only the main artery, but every patent arteriole throughout the mass, and causes the whole tense mass to vibrate.'

"Von Frey's explanation of the fact that when the arm is plunged vertically into a dish of mercury the shock of the pulse can be felt with special distinctness at a very definite point, is of interest in this

connection. He is of the opinion (7) that the pulse is felt most distinctly at that place because there the blood flow is checked, the pulse waves are reflected positively, and the summation that results gives to the waves sufficient energy to produce a sensible pulsation. It may, however, be said now that this explanation cannot be the correct one, for if it were, the sensation caused by simply occluding the artery should be as intense as the sensation that is experienced when the compressing pressures lies between the systolic and diastolic level. As is well known, this is not the case.

"Gittings (8), while accepting the view with regard to sound production prevailing at the time he writes (1910), believes that the compression chamber contributes to the sound by virtue of its action as a resonator. This conclusion he bases upon the observation that the sounds are much louder when the artery is compressed with the usual pneumatic cuff than with an Esmarch bandage.

THE NEW HYPOTHESIS

"The investigation of arterial sounds which forms the basis of this paper forces us to the conclusion that the main mechanism of the compression sounds is as follows: Under compressions which permit the pulse to determine relatively wide excursions of the arterial wall in the compression chamber, that is, under compressing pressure ranging from systolic arterial pressure to, and even a variable distance below, diastolic pressure, the volume of the compressed artery increases abruptly with each pulse (1). This permits a considerable volume of blood to enter the opening artery with a high velocity. The motion of this column of blood is, however, suddenly checked where it comes into contact with the stationary, or practically stationary, column of blood filling the uncompressed artery below. The water hammer that is thus set into play distends the arterial wall at the point of impact with unusual violence. This distention sets the arterial wall into vibration and the sound is produced."

Against the water-hammer hypothesis of Erlanger the following objections may be raised.

Erlanger and others have found that the first sound may be heard over the distal segment before there is the slightest record of any pressure or volume change in the distal segment as indicated by a sphygmo-

gram. The latter does not begin to record any pulse changes until the pressure in the cuff is lowered an additional 10 mm. of Hg. The reduction must be even greater before the pulse changes assume any considerable size. (See Erlanger, fig. 2, p. 104.)

Regarding the site of formation of the sound, MacWilliam and Melvin (3) report as follows (p. 184). "When using an artery in the compression tube in the way described, with no piece of artery on the distal side, but the phonendoscope or tambour applied over the glass tube issuing from the artery in the compression tube, we find that the sounds are perfectly well developed and characteristic—a fact which controverts the idea of the essential importance of the artery distally to the compressed area."

We might add further that the effect of water hammer in the blood stream can reach a magnitude worthy of notice only when the arterial wall is distended and rigid, and when the arrest of the blood flow is sudden and complete. Water hammer, such as it is, plays a rôle only when the arterial flow is more or less completely obstructed by the cuff, and its effect is felt proximal to the point of construction.

The present writer wishes to offer an explanation of the method of production of these sounds, based on an analysis of the various factors concerned, their change during decompression of the cuff, and their effect upon each other. The validity of such an analysis can be accepted as complete only if it is capable of explaining not merely the experimental findings of previous workers but also the many clinical anomalies which thus far have not been clarified.

Sounds are *produced* by bodies which have been made to vibrate by the application of some force. The quality of the sound depends upon the nature of the sounding body, *i.e.*, the shape, the substance of which it is made, etc. The clear pistol-shot sound emanating from an arterial wall which has been made to vibrate differs in quality from the murmur arising in blood which is vibrating because of forces applied to it in the form of resistance due to constriction of the arterial channel, sudden widening, sudden change of direction, etc. Over the ante-cubital fossa during decompression of the cuff, the first clear sound which continues throughout the first phase, and which usually becomes so loud in the third, to gradually fade off in the fourth, arises in the arterial wall, and the intensity of the sound in each phase varies

with the amplitude of vibration of the wall. The murmur which is so prominent in the second phase arises during the rapid flow of a considerable amount of blood through a partially constricted artery.

A sound will arise in the arterial wall when it is made to vibrate by sudden distention. The intensity of the sound will depend upon the amplitude of vibration, which in turn is determined by the magnitude

FIG. 1

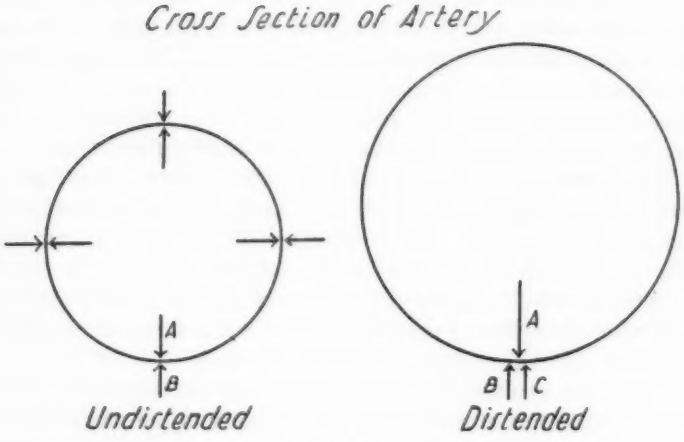

FIG. 2

of the distending force, the suddenness with which it is applied, and the initial state of tension of the arterial wall. The loudest sound will occur when the arterial wall is suddenly stretched from a condition of rest, or zero tension, by a large distending force.

Consider a segment of the brachial artery about 10 cm. long open at both ends and containing air (Fig. 1). The normal artery, although soft and distensible, has sufficient rigidity to resist the force of gravity which draws down the upper part of the artery and tends to flatten

the circular lumen. In the normal artery, however, both the rigidity of the resting wall, and the force of gravity acting on the mass of the arterial wall, are small enough to be neglected. In the sclerotic artery, the initial rigidity of the artery at rest may prove large enough to merit consideration.

The segment illustrated, open at both ends and containing air, will have a cross-section that is approximately circular, the size of the circle depending upon the size of the artery in the undistended or resting condition. Ordinarily one says that the artery is at rest because the intra-arterial pressure is zero. Strictly speaking, the arterial wall is at rest because the forces acting upon it are in equilibrium with each other. The pressure inside the artery is equal to atmospheric and tends to distend the wall with a pressure head of 760 mm. of Hg. The pressure outside the artery is also atmospheric, and tends to compress the artery with a pressure head of 760 mm. of Hg. The arterial wall is at rest and under zero tension because the force which is tending to distend it is exactly equal to and counterbalanced by the force which is tending to compress it. If the intra-arterial pressure is reduced below the extra-arterial, the artery will immediately collapse. If the intra-arterial pressure rises above the extra-arterial, the artery will be distended, and the amount of distension will increase as the excess of intra-arterial over extra-arterial pressure increases. For equal increments of intra-arterial pressure, extra-arterial pressure remaining the same, the amount of distension with each increment will be steadily diminished. After the artery has been distended to any given point the system is still in equilibrium, but a new force is now acting. This new force is due to the acquired state of tension of the artery, by virtue of which the artery tends to compress the blood it contains, and the amount of this pressure is now equal to the excess of intra- over extra-arterial pressure, the latter being 760 mm. of mercury.

A equals total intra-arterial pressure (*i.e.*, atmospheric plus increment.)

B equals atmospheric pressure.

C equals pressure due to state of tension of the arterial wall.

These are the only three forces acting and under all circumstances.

A equals B plus C

When the extra-arterial pressure is equal to the intra-arterial, A equals B, and C equals O; *i.e.*, the arterial wall is at rest.

C equals A minus B

The amount of tension is equal to the excess of total intra-arterial over extra-arterial (atmospheric) pressure.

The actual amount of distention of the artery depends not merely on the size of the distending force but also upon the height of the extra-arterial pressure. The artery cannot be distended without decreasing the volume of the air in the pneumatic constrictor, and the higher the pressure in the constrictor the more will it oppose further compression.

When an artery through which blood is flowing is compressed from the outside, the pressure necessary to close the artery to the point of complete flattening is equal to the sum of four factors which are tending to keep the artery open or to open it, after it has been compressed. These factors are: first, the inherent stiffness of the arterial wall, which in the normal non-sclerotic brachial artery of man is very small. Secondly, there is the pressure head of the blood in the artery. Thirdly, there is the velocity head which adds itself to the pressure head, because the kinetic energy due to forward motion of the blood has been converted into potential energy by obstruction to the flow. The fourth factor is the intra-arterial pressure due to the effect of water hammer; this factor comes into play, like the third, only when the flow is obstructed, and depends upon the length of the column of blood that is obstructed, upon the velocity of flow at the time of obstruction, and upon the suddenness with which the flow is stopped.

Of these four factors the first, stiffness of the arterial wall, may be neglected for the normal brachial artery because it does not amount to more than 2 mm. of Hg. The second factor, pressure head, is by far the most important under ordinary circumstances, and may be measured in mm. of Hg. by means of a piezometer tube or by taking the maximal pressure in a branch artery which comes off at a right angle. The velocity head cannot be measured because we do not know the maximum velocity of flow, and for the same and further reasons, the effect of water hammer cannot be measured at present. We need not be discouraged, however, because the sum of factors 3 and

4 may be obtained by subtracting factor 2 from the total, realizing that factor 1 is small enough to be neglected.

Figures taken from Dawson (4) will provide a rough estimate of the sum of factors 3 and 4.

It is apparent that the maximal pressure in the carotid artery is equal to the sum of factors 2, 3, and 4. The maximal pressure in the thyroid acting as a piezometer is equal to factor 2. The difference is equal to 3 plus 4.

Left carotid	160	Right carotid	154
Left thyroid	140	Right thyroid	140
	20		14

It is of course hazardous to compare conditions in the carotid artery of an anaesthetized dog with those in the brachial of a normal man. For the time being we must content ourselves with approximations. For various reasons, we are inclined to believe that 14 mm. of Hg. gives one a fairly good estimate of the pressure due to the sum of velocity head plus the effect of water hammer, when the brachial artery of man is obstructed. When the flow of a steady stream is slowly stopped, the velocity head is completely converted into pressure head, while the effect of water hammer is practically nil. When the arrest of the flow is sudden, water hammer plays a more important part. We cannot at this time discuss the question of water hammer. For our purposes we must bear in mind that water hammer is increased by increased velocity and that if there is little or no obstruction to the flow of blood through the artery, water hammer plays no rôle whatever, while velocity head continues in the form of kinetic energy of the moving blood, without causing any increase in the pressure head. Indeed, when there is cone-like constriction of an artery, with increased velocity of flow where the artery is narrowest, the pressure head in the constricted portion is likely to be reduced in accordance with Bernouilli's principle.

In order to understand the mechanism of production of the Korotkoff sounds, we must trace the course of the progressive changes, which take place during decompression, first in the extra-arterial pressure; secondly in the tension of the arterial wall; and thirdly in the intra-arterial pressure. During decompression there is also a change in the size and shape of the arterial lumen. We shall trace the pro-

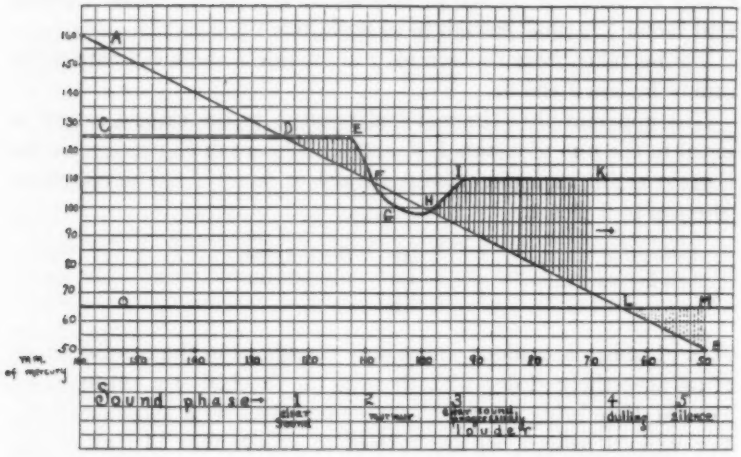

FIG. 3. DIAGRAM TO SHOW RELATIONSHIP BETWEEN PRESSURE CHANGES AND SOUND PRODUCTION

Line AB indicates extra-arterial pressure, *i.e.*, pressure in the cuff during decompression.

Line CEFGHIK indicates the maximal systolic pressure head in the artery proximal to and beneath the cuff.

Line OM indicates the intra-arterial diastolic pressure head.

Vertical continuous lines indicate excess of systolic intra-arterial pressure head over extra-arterial pressure. This is a measure of the distending or sound-producing force.

Vertical interrupted lines indicate excess of diastolic intra-arterial pressure head over extra-arterial pressure. This measures the force which keeps the artery distended during diastole, thus minimizing the further distention or sound production that might result from a systolic increase of intra-arterial pressure head.

The line CEGIK is correctly placed in relation to AB, but its assigned level and the form of the curve EGI are probably only approximately correct.

Upon correlating the sound phases with the pressure changes one sees that the intensity of the sound varies with the force producing it (Phases 1 and 3).

In the second phase the sound-producing force is decreased, then absent. The murmur heard during this phase arises from the passage of blood under pressure through a constricted orifice. (See text for more detailed discussion.)

gressive changes of these various factors and see how they explain the first appearance of the sound, and the changes in quality and intensity which the sound undergoes.

The force which tends to distend the artery beyond its resting position is equal to the excess of intra-arterial systolic pressure head over extra-arterial pressure. The amplitude of the excursion is limited, first, by the tendency of the artery to resist stretching, which from the resting position is not great, and secondly, by the pressure already existing in the cuff; volume increase in the artery must be accompanied by volume decrease in the cuff, consequently, by pressure increase of the cuff, as is indicated by the sudden rise of the mercury column as the compressed artery is partly filled during systole. With the pressure in the cuff at 75 mm. of mercury, the resistance to compression, *i.e.*, to distention of the artery, is not so great as it is at 150 mm. of Hg., and neglecting the tension of the artery, a given excess of intra-arterial over extra-arterial pressure will distend the artery farther when the cuff is at 75 mm. than when it is at 150 mm. of Hg. The greater distention will increase the intensity of the sound, which varies with the amplitude of vibration.

In Figure 3 as described in the accompanying legend we have attempted to present a graphic record of the continuous change in the physical factors concerned. The sound phases are also charted.

Line AB represents the pressure in the cuff during decompression, the value being indicated on both ordinate and abscissa. Line CDEFGHIK represents the maximal systolic intra-arterial pressure head at the proximal end or cone-like portion of the compressed artery when the latter is obliterated, or in the lumen of the compressed artery when the latter actually opens to permit passage of blood.

In general the position of this line on the chart is correctly placed in relation to line AB, but the exact level of the line, as well as the shape of the curve EGI, are only approximately correct but will serve for qualitative considerations.

The height of the line CD representing the maximal systolic pressure head when the artery is completely obliterated, is a measure of the sum of

1. Systolic pressure head
2. Systolic velocity head
3. Increment due to water hammer

The force tending to distend the artery equals the excess of that sum ever the extra-arterial pressure and that excess is graphically represented by the continuous vertical lines. It increases from D to E, causing the sound to become louder. Not only is the distending force increasing during this phase, but the extra-arterial pressure which opposes distention of the artery decreases, thus augmenting the effect of the distending force in causing the arterial wall to vibrate.

Phase 2 at point E, is characterized by the appearance of the murmur and dulling or disappearance of the sound. The appearance of the murmur indicates that for the first time during decompression a considerable amount of blood is passing beneath the cuff. This blood, during the continuation of the murmur at least, passes through a partially compressed artery at a very rapid rate into the distal arterial segment.

The beginning of phase 2 is also characterized by certain important intra-arterial pressure changes. When the cuff is no longer capable of keeping the artery compressed, the arterial lumen is more and more widely opened during successive systoles, while the extra-arterial pressure is falling. The opening of artery even below its normal resting diameter practically removes the effect of water hammer, and prevents the conversion of velocity head into pressure head. Indeed, since the velocity head of the blood passing through the constricted portion is higher than in the proximal wide segment, the pressure head in the former must be considerably less than in the latter portion (Bernoulli's Principle).

The murmur in phase 2 indicates that the artery is partially compressed which proves that intra-arterial systolic pressure (G) has fallen below extra-arterial pressure.

As the pressure in the cuff falls from F to H, the artery is less compressed; at H it assumes its resting diameter, because at H intra-arterial pressure equals extra-arterial pressure and the murmur disappears at that level or shortly thereafter.

At the beginning of phase 3, once more the intra-arterial pressure exceeds the extra-arterial, the excess gradually increasing from H to I to K, the sounds becoming louder and louder, not merely because of the continuing increase in the excess, but also because the extra-arterial pressure is falling and permits more stretching of the arterial wall, for a given stretching force.

Assuming that OM indicates the intra-arterial diastolic pressure head, the sounds will continue to increase in intensity until point L is reached. Here the intra-arterial diastolic head equals and begins to exceed the extra-arterial pressure. From this point on, the arterial wall assumes a state of higher and higher tension during diastole. The higher the initial tension of the arterial wall, the less effective will be a given force in further distending it. Hence, after point L has been reached, the sounds will diminish in intensity more or less rapidly, although such diminution will be slightly prolonged by the falling extra-arterial pressure which encourages arterial distention.

Assuming that we understand in a general way how the sounds are produced, we might pertinently ask, "What do they indicate?"

The first appearance of the sound *does not* indicate normal intra-arterial systolic pressure head. The sound appears while the artery is still obliterated and the distending force, which is tending to produce the sound and which is opposed by the extra-arterial pressure, is equal to the total head in the proximal segment, which is equal to the sum of pressure head, velocity head, and increment due to water hammer. The sum of the last two factors in the carotid artery of a dog is in the neighborhood of 14 mm. of Hg.

The dulling of the fourth sound is due to the fact that the arterial wall has taken on a certain amount of tension during diastole, and the efficacy of the increase of tension during systole in producing a sound is mitigated by the initial state of tension of the artery. Just how great this initial state of tension must be is yet to be determined, but for the present it seems reasonable to take the beginning of the fourth phase as an index of intra-arterial diastolic pressure head.

Without the use of a pressure cuff, one fails to hear any sound over the brachial artery of the normal person, the reason being that during diastole the wall is in a state of considerable tension or in a condition of considerable distention, and the systolic increase of pressure head is insufficient to produce a sound. Where the diastolic distention is reduced, and where the systolic increase of pressure head is considerable or sudden, sounds may be set up in the arterial wall in the absence of a compression cuff. These latter conditions are fulfilled wherever the pulse is collapsing, and the sounds are exceedingly loud in aortic insufficiency, *i.e.*, the so-called pistol-shot sound heard over

the arteries. The sounds are produced by vibrations resulting from sudden distention of the arterial wall, and are evidence of actual mechanical strain. The writer cannot help suspecting that a collapsing pulse at least predisposes to some anatomical change in the arterial walls, but this still remains to be proved.

With this analysis of the mechanism of production of the Korotkoff sounds, the earlier experiments are not difficult to understand.

Korotkoff succeeded in producing a sound by pouring salt solution into the iliac artery of an animal. The sound, of course, is produced by the sudden distention of an artery previously at rest.

MacWilliam and Melvin found that the "sounds are perfectly well developed and characteristic" when the *artery* consists merely of a tube in a compression chamber. "The sounds . . . . owe their origin to vibration of the arterial wall when the normal circular form of the vessel is, in the compression area, more or less distorted by external pressure." With regard to the location and origin of the sound, we agree entirely with MacWilliam and Melvin. So far as we can see, however, actual distortion of the artery is not necessary; merely a state of rest or reduced tension during diastole is sufficient, as illustrated in the arterial sounds in aortic insufficiency or other conditions where the pulse is collapsing.

Von Frey's experiment is interesting. He found that "when the arm is plunged vertically into a dish of mercury the shock of the pulse can be felt with special distinctness at a very definite point." Von Frey's explanation and Erlanger's objection are cited above. As we understand it, the shock of the pulse can be felt when the extra-arterial pressure exerted by the mercury is sufficient to place the arterial wall at rest during diastole, the shock being felt by the sudden distention during the next systole.

Gittings' observation that the sounds are much louder when the artery is compressed with the usual pneumatic cuff than with an Esmarch bandage, depends, as we see it, upon the fact that both serve equally well in placing the artery at rest during diastole, but that the unyielding Esmarch bandage practically precludes arterial distention during systole.

A phenomenon of considerable interest is the so-called auscultatory gap, which is a period during decompression when no sounds are heard.

The literature is reviewed and thirty cases are described by S. G. Mudd and P. D. White (5). The auscultatory gap is of interest in this connection because it calls for an explanation and puts to the test analysis which we have made of the mechanism of production of the Korotkoff sounds.

Mudd and White point out that in their series of 30 patients, hypertension, aortic stenosis, or both occurred in every case and that

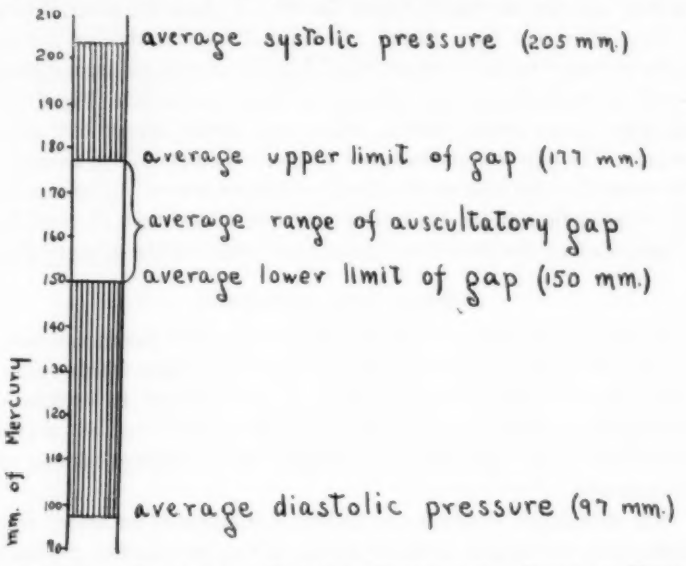

FIG. 4. AVERAGE SYSTOLIC AND DIASTOLIC PRESSURES IN A SERIES OF THIRTY CASES, SHOWING THE AVERAGE RANGE AND POSITION OF THE AUSCULTATORY GAP

(From Mudd and White)

"the auscultatory hole in hypertensive cases may be related to a type of pulse wave showing slight anacrotism or plateau form. The gap that occurs in aortic stenosis is probably due to reduced velocity of the pulse wave through the arteries, associated with rising or plateau pulses, some of which show anacrotism. Reduced velocity of the wave accompanied by a slowly rising pulse may also furnish an expla-

nation for the auscultatory gap when it is dependent on an aneurysm or compression of the subclavian or brachial artery."

Just as the collapsing pulse with its sudden rise during systole and its low diastolic pressure head is the most effective for producing an arterial sound, so is the plateau pulse with its slow rise and its high diastolic pressure head the least effective in the production of a sound.

Concerning the mechanism of production of the auscultatory gap, we may say that normally during the second phase the arterial sound is first dulled and then absent, but the dulling and absence are normally obscured by the murmur which appears during the second phase. It will be noticed from the diagram of Mudd and White (Fig. 4) that the gap occurs approximately where one would expect the second phase. The gap is abnormal, not so much because of the absence of the sound but because of the absence of the murmur. The murmur, we believe, disappears because of the diminished rate of *blood flow* as indicated by the slow rise of the anacrotic limb of the sphygmogram.

SUMMARY AND CONCLUSIONS

An analysis is offered of the factors concerned in the mechanism of production of the Korotkoff sounds and the changes which occur in these factors during decompression, in an attempt to explain not merely the manner in which these sounds are produced but also the qualitative and quantitative changes they undergo during decompression.

The sounds are produced by sudden distention of an artery whose walls were previously relaxed because of the surrounding pneumatic cuff whose pressure opposes the diastolic intra-arterial pressure and keeps the artery undistended during diastole.

The appearance of the first sound is not a measure of systolic pressure head, but rather of the sum of pressure head, velocity head, and such increment as may be due to water hammer.

The dulling of the sound at the beginning of the fourth phase probably occurs just below the intra-arterial diastolic pressure head.

During the second phase the arterial sound weakens or disappears and is supplemented or replaced by a murmur. In such cases of hypertension or aortic stenosis where the rate of arterial blood flow is not rapid enough to produce a murmur during the second phase,

there is produced an auscultatory gap, since there is nothing to supplement or replace the arterial sound which during that phase weakens or disappears.

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TOTAL EXTIRPATION OF THE DOG'S LIVER IN ONE STAGE[1]

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From the Surgical Department of the Johns Hopkins Medical School with the aid of a grant from the Hartley Corporation

Early in 1928 our interest in the question of the total extirpation of the dog's liver was aroused by Dr. E. M. K. Geiling. A review of the methods described showed that they all required a two-stage or a three-stage operation. The interim between the stages was from 2 to 10 weeks. Indeed, it had been stated that complete removal of the liver of a dog in one stage is practically impossible.[2] Two questions arose in our minds: (1) Is it not possible to devise a one-stage operation; and (2) Can accurate deductions be drawn from the removal of a liver that has been deprived of its portal circulation for several weeks? The effort to answer the first question led us to evolve the technic which we shall describe later. The answer to the second question should not be made without painstaking comparisons between the results obtained by the use of our technic and by the other operative methods. We have, however, obtained suggestive data which will be set forth in a subsequent paper.

The basic idea underlying our operative technic is the shunting of the portal and vena cava circulations through a glass cannula before removing the liver. There are two fundamental principles involved in the execution of this operation. The first is that an animal will tolerate the simultaneous interruption of both the portal vein and the abdominal vena cava for many minutes. In an animal under very light ether anesthesia, we clamped off the hepatic artery, the portal vein, and the abominal vena cava. After 31 minutes had elapsed the clamps were removed, the circulation was spontaneously resumed, and 20 minutes later there was no discernible local or systemic effect. In none of our extirpation cases have we found it necessary to suspend the

[1] Received for publication October 3, 1928.

[2] Mann, F. C.: *Medicine*, 1927, (December), vi, 419.

circulation for more than 13 minutes. The second principle is that dog's blood does not clot within 24 hours when passing through a cannula made of Pyrex glass.

It would be interesting to state the various procedures and modifications which were attempted before arriving at our present technic. Such an account, however, would serve no real purpose, and might confuse the reader.

Choice of animal. A medium-sized, healthy dog is preferable. By medium-sized we mean a dog weighing between 7 and 10 kgm. Those with long bodies and shallow chests are best. It is also preferable to have animals that have not been caged up for any length of time. Ordinarily, collies, poodles, and airedales are poorer risks than fox-terriers and bulldogs.

Preparation and anesthetic. The only preparation is the omission of feedings for 18 hours before operation. This is merely to insure an empty stomach. Morphine as a preliminary hypodermic is avoided because of the stupor it induces for 3 to 4 hours after the operation. We found that the very best results were obtained with a light ether anesthesia given by the open method. In an effort to secure an anesthetic which would not alter the blood sugar we tried amytal intravenously and procaine intraspinally. Neither of these was very successful.

Operative technic. The operative technic is as follows: A long right rectus incision is made 1 cm. from the midline; at the upper end this is curved mesially to the xiphoid (Fig. 1, *a*). The peritoneal fold between the liver and the right kidney is divided. The vena cava just above the lumbo-adrenal veins is then mobilized and encircled with a ligature of braided silk (Fig. 1, *b* and *c*). The omental attachments to the liver are divided between clamps and ligated with fine silk. The hepatic artery and the common bile-duct are isolated, clamped, divided, and ligated (Fig. 1, *d*). The portal vein and the vena cava are then the only remaining structures attached to the lower portion of the liver. The liver is then retracted from the diaphragm and the thin avascular suspensory ligaments are divided (Fig. 2, *a*). Care is taken not to damage the diaphragmatic veins entering the vena cava at the upper margin of the liver. The stomach is then retracted downward and to the left, and with blunt dissection the peritoneal reflection along the left posterior margin of the liver is divided. With the

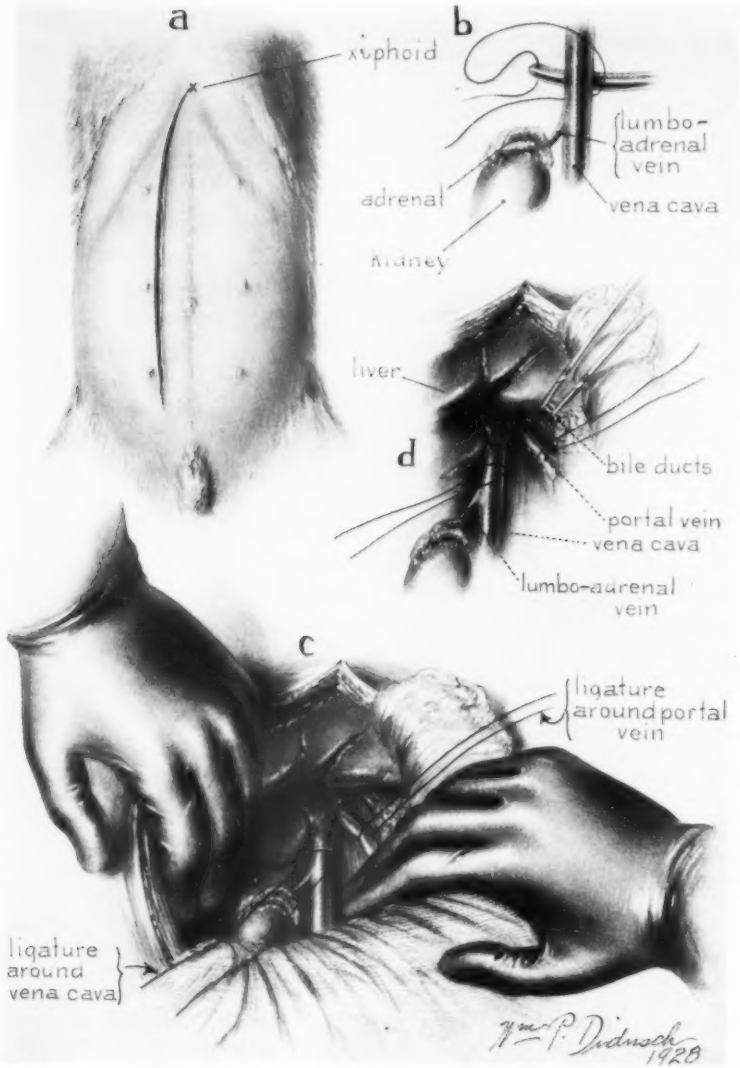
a
xiphoid
b
lumbo-adrenal vein
adrenal
vena cava
kidney
liver
d
bile ducts
portal vein
vena cava
lumbo-adrenal vein
c
ligature around portal vein
ligature around vena cava
Wm. P. Didusch 1928

FIG. 1

index finger the liver and the vena cava are raised and separated from their posterior attachments. The operator then perforates the peritoneum along the right posterior lateral border of the liver (Fig. 2, *b*), thus producing a communication with the space that has just

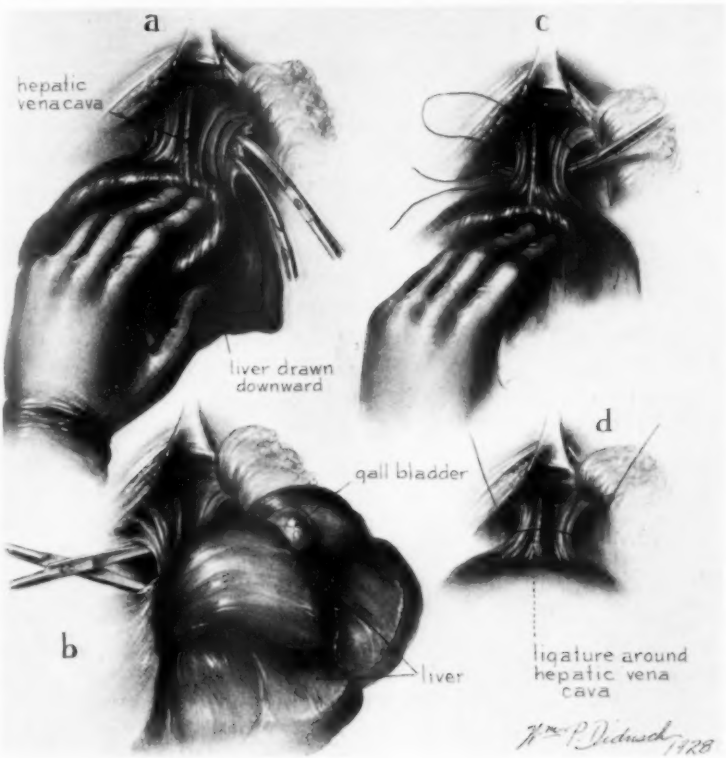

FIG. 2

been made. A curved clamp is passed through this tunnel under the liver, and a heavy braided silk ligature is drawn through the opening. This ligature is then passed around the lobes of the liver and brought to lie on its upper surface. One end of the ligature is again passed through the tunnel and brought into place between the diaphragm and

the liver (Fig. 2, *c* and *d*). This makes a complete loop about the hepatic vena cava. For the sake of clarity this will be designated the diaphragmatic ligature. Ordinarily, no bleeding is encountered in this manoeuvre. The field of activity then shifts to the region of the portal vein and the vena cava; the liver is stripped off the vena cava until the first hepatic branch is fully exposed. Anteriorly this is accomplished with the handle of the scalpel, and posteriorly by drawing from side to side the ligature previously placed about the vena cava. If too much bleeding occurs, further stripping is postponed until the circulation has been cut off (Fig. 3, *b*).

Up to this time there has been no necessity for haste. With the next few procedures, during which the portal and vena cava circulations are suspended, it is desirable to waste no time. The portal vein is doubly clamped *just as close to the liver as possible*. This is not quite correctly shown in Fig. 3, *a* and *b*. The Kelly clamp should be closer to the liver and the mosquito forceps just as close to the Kelly clamp as is practicable. The vein is then divided between these clamps and the hepatic end ligated (Fig. 3, *a* and *b*). The vena cava is clamped between the braided silk ligature and the right lumbo-adrenal vein (Fig. 3, *c*); at the same time firm traction is made on the loop and the ends of the diaphragmatic ligature. This latter manoeuvre successfully prevents back bleeding. The vena cava is then incised longitudinally at a point about 1.5 cm. above the right lumbo-adrenal vein (Plate 3, *c*). The incision is carried up into the liver for a distance of 2 to 3 cm. A properly prepared cannula (Fig. 3, *d*) filled with salt solution is slipped into the open vessel and passed upward until it meets the obstruction caused by the taut diaphragmatic ligature. This is momentarily relaxed and the cannula pushed beyond it (Fig. 3, *e*). Firm traction upon the ends of the ligature serves to draw the encircling loop tightly around the end of the cannula lying within the hepatic vena cava. The loop effectually prevents bleeding around the cannula and also helps to hold it in place. The lower ends of the cannula are fitted with cork or rubber stoppers to prevent hemorrhage. A Carrel or Kelly clamp is applied 1 or 2 cm. from the severed end of portal vein and this end opened by removing the mosquito artery forceps. At the time that the drawings were being made, we were applying the Carrel clamp and the ligature earlier than is necessary.

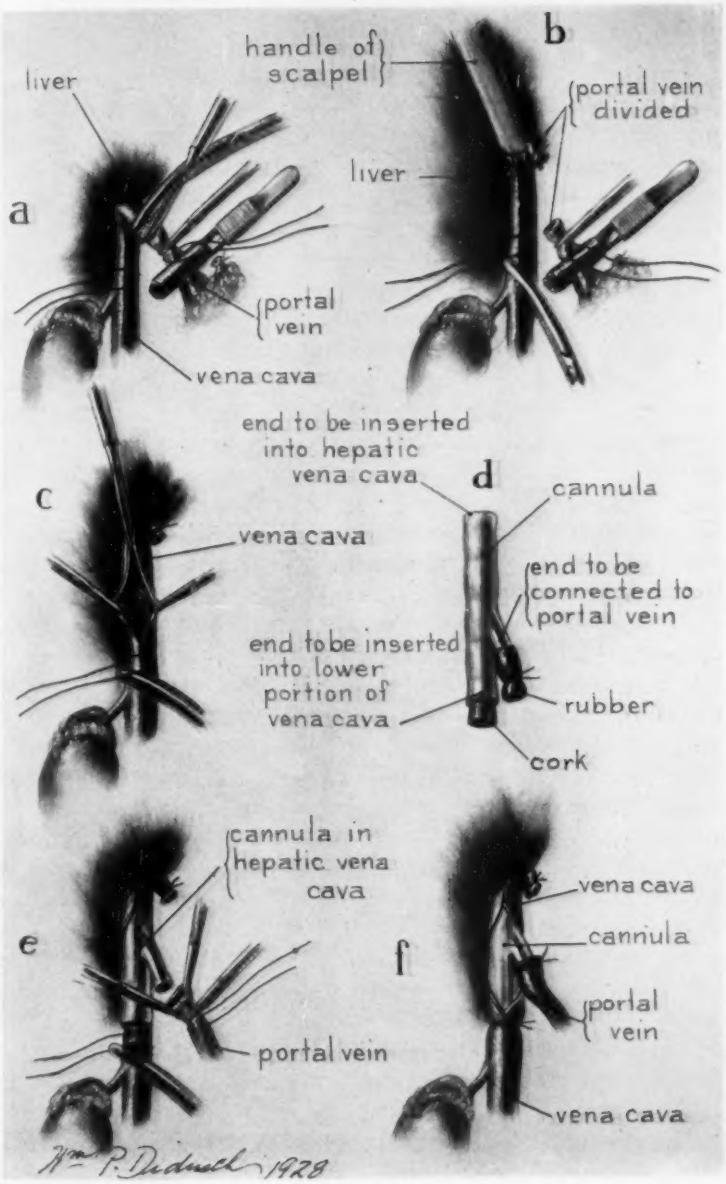
a
liver
portal vein
vena cava
b
handle of scalpel
liver
portal vein divided
c
vena cava
d
end to be inserted into hepatic vena cava
cannula
end to be connected to portal vein
end to be inserted into lower portion of vena cava
rubber
cork
e
cannula in hepatic vena cava
portal vein
f
vena cava
cannula
portal vein
vena cava
Wm P. Didusch 1928

FIG. 3

A ligature with one tie in it is placed about the open end of the vessel and the margin of the divided end is grasped with three delicate forceps and kept patent, while the portal arm of the cannula is inserted and the ligature tied (Fig. 3, *e*). The Carrel clamp is removed, and immediately the portal blood flows through the cannula in the vena cava. While the assistant is placing the three forceps on the mouth of the portal vein, the operator removes the rubber plug from the portal end of the cannula. The alternate aspiration of air and the discharge of blood is prevented by the operator holding his finger over the opening of the cannula until it is ready to be shoved into the mouth of the portal vein. The operator then forces the cannula upward toward the diaphragm, and often has to extend the incision in the hepatic vena cava to prevent the portal arm from retarding the upward passage of the cannula. The cork is then removed from the lower end of the cannula, and, with a finger over the open end, the cannula is slipped into place in the vena cava. To facilitate this procedure, the edges of the incised vena cava are held apart with clamps. The previously placed ligature is carefully pushed upward so as to engage the Marie at the lower end of the cannula. The loop is then drawn taut and the clamp removed, thus completely restoring the circulation (Fig. 3, *f*).

There is always some loss of blood (5 to 50 cc.) as the lower end of the cannula is being slipped into place. This blood is removed and the position of the ligature inspected before it is tied.

Once the circulation is shunted through the cannula the diaphragmatic ligature is inspected and tied securely. The liver is then removed (Fig. 4, *a*). We usually do this by blunt and sharp dissection, beginning from above and working downward. Nearly always the liver comes out *in toto*. Care is taken to leave a posterior strip of the hepatic vena cava (Fig. 4, *b*). Inspection is then made to assure the operator that there is no kinking of the portal vein and that all bleeding is checked. Sometimes it is necessary to pass a mattress suture posterior to the hepatic vena cava in order to tie off small tributaries. As soon as the operator is satisfied that the circulation is passing properly through the cannula the abdomen is closed with silk, layer by layer.

The ether mask is always removed when the portal circulation is reëstablished. This assures us of a conscious animal when the opera-

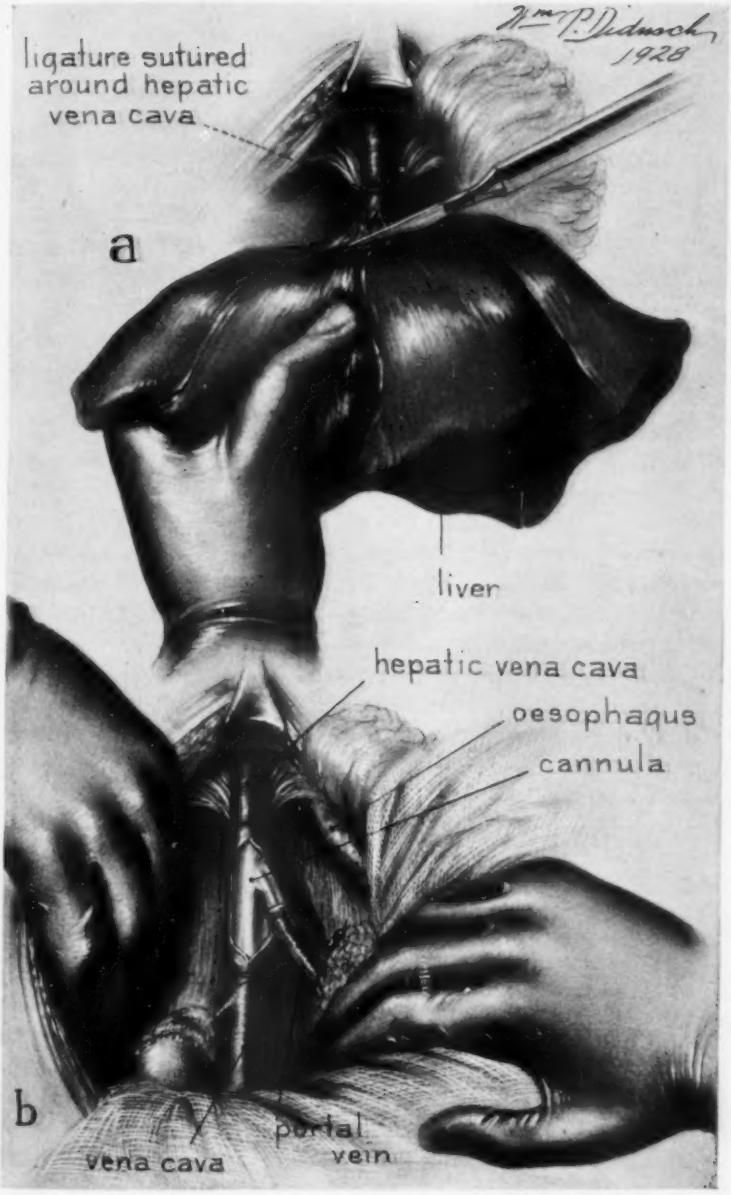
Wm P. Didusch
1928
ligature sutured
around hepatic
vena cava
a
liver
hepatic vena cava
oesophagus
cannula
b
vena cava
portal
vein

FIG. 4

tion is ended. The time required for the whole procedure varies. On several occasions we have done the complete operation in less than 40 minutes. On two occasions the skin was closed 35 minutes after the incision had been made.

The time elapsing between the clamping-off of the circulation and its reëstablishment varies with the dexterity of the operator and the anatomical variations in the organs. Our fastest time for doing this part of the operation is 5.5 minutes. In this case the portal blood was stopped only 3.5 minutes. In one dog the portal circulation was cut off 7.5 minutes and the vena cava 12.5 minutes without apparent untoward effects, for this particular animal lived over 15 hours.

Discussion. It might prove instructive to enumerate some of the difficulties which we have encountered and to consider briefly the commoner causes of failure.

In the beginning we feared that clotting in the cannula would prove an effectual drawback to this procedure. We gave, therefore, appropriate amounts of heparin or novhirudin. We have found, however, this precaution to be entirely superfluous, for the flowing blood will not clot in Pyrex tubes under 24 hours.

Considerable difficulty was experienced with the hepatic end of the vena cava. For a while we opened the diaphragm to place a ligature about the thoracic end of the vessel. Passing a braided silk ligature under the liver and then looping it about the hepatic portion of this vessel is a far superior method. In handling this ligature we have found the following manoeuvre to be of real service. It consists in drawing the loop down so that it ties loosely about the vessel but does not constrict it. This is shown in Fig. 2, *d*. The center of the loop is then caught with the tip of a curved clamp, and this remains attached while the portal vein is being divided and the cannula inserted. Traction upon the ends of the ligature and on the curved clamp effectively prevents back bleeding while the vena cava is open. As soon as the end of the cannula passes the diaphragmatic ligature, the clamp is removed and the ends of the ligature are drawn taut. The loop is already in place, and consequently a minimum of time and of blood is lost.

Another barrier to success was the shortness of the vena cava between the right lumbo-adrenal vein and the liver. This was overcome by stripping the liver tissues from the vessel.

A very considerable difficulty was encountered in the kinking of the portal vein. To overcome this it was necessary to set the portal arm of the cannula far back so as to keep this vessel on a stretch. Sometimes the portal vein would tear before or perforate after being connected with the cannula. The tearing usually came from the ligature being placed around the vessel too long before it was tied; consequently, we postponed putting this thread of silk about the portal vein until the moment before the cannula was inserted. The perforations can best be avoided by refraining from too much dissection of the vessel wall and also by avoidance of crushing the vessel distal to the point of ligature. In a few animals we found an accessory portal vein running from the duodenum to the liver. It was impossible to connect this with the cannula, and gangrene of the upper bowel was threatened. At various times, when inserting the cannula, we were annoyed because it would slip away from us. We had a great deal of difficulty in holding the cannula securely and accurately, until we chanced upon the idea of fastening a ligature of braided silk about it at the level of the portal arm. This affords a splendid handle to an otherwise slippery and elusive tube.

We find it an advantage to have on hand cannulae of different sizes, in order to have one suitable for the size of the dog. The measurements of an average cannula are: length 8.5 cm.; bore 10 mm.; portal arm 3.5 cm. from lower end of cannula; bore of portal arm 7 mm.

The commonest cause of failure is hemorrhage from one source or another during the operation. Awkwardness in threading the portal arm into the portal vein is our most frequent drawback. This vein is a delicate structure and tears easily. On two occasions our cannula perforated the hepatic vena cava instead of entering it.

On three or four occasions the animals at autopsy showed a small amount of air in the chambers of the heart, but this did not seem sufficient to cause their death. Edema of the diaphragm occurred at first from occlusion of the diaphragmatic veins by the ligature around the upper end of the cannula. A little care, however, prevents the inclusion of these veins.

Too much emphasis cannot be laid upon the necessity of a light anesthesia. We endeavor to stop giving ether at the time the circulation is cut off, and always remove the mask by the time the circulation is restored.

Observations. Our primary object has been to devise a reliable and simple method for the removal of the liver in a single operation. In the course of this endeavor we have made certain observations which are instructive.

A high percentage of success (80 to 90 per cent) can be expected after one becomes familiar with the operative procedure. Secondly, the time required varies from 35 to 50 minutes. Thirdly, the animals recover sufficiently to walk, bark, run around, void, defecate, and drink water. If untreated they will all die within 2 to 3 hours after the liver has been removed. Death is associated with symptoms of hypoglycemia.

Fourthly, if given glucose intravenously, they live from 10 to 16 hours. Their death at that time is one of gradual cardiac failure preceded by vomiting. The actual cause of death is not clear, but most of the experiments were carried on in excessively hot weather, and this surely tends to be a lethal factor.

It is a pleasure to acknowledge our appreciation of the valuable assistance of Dr. Ed. J. Poth in making the cannulae, and of the efficient anesthesias given by Mr. M. J. Ullrich.

HEART BLOCK DUE TO PRIMARY LYMPHANGIO-ENDOTHELIOMA OF ATRIO-VENTRICULAR NODE

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In 1911 Armstrong and Mönckeberg (1) described in detail a case of intermittent complete heart block in a five and one-half year old boy due to a primary tumor of the atrio-ventricular node infiltrating the bundle of His. This was said to be the first tumor of its kind to be reported. In 1924, Mönckeberg (2) referred to the same case and stated that in the intervening period no similar tumors had been described. Recently in this laboratory an autopsy has revealed a tumor practically identical with that described by Mönckeberg, both in form and in location; it is recorded here because of its pathological as well as its clinical interest.

A review of the literature has brought to light no other heart tumors having the same characteristics as these, which apparently belong to the group of lymphangio-endotheliomata. Weber (3) has reported two cases of cavernous varicosities projecting into the right auricle in the region of the foramen ovale, but these, he states, communicate with veins and are probably not neoplastic in character. Penneman (4) has described a hemangio-endothelioma of the heart, and Manifold (5) a cavernous angioma in the wall of the left auricle, neither of which involves the conductive mechanism, or bears any close relation to the case at hand. An extensive sarcoma of the heart infiltrating the entire septum and interfering with the His bundle has been described by Martin and Klotz (6), and countless secondary tumor metastases involving some portion of the conductive apparatus are to be found in the literature. Of primary tumors actually arising at the site of the atrio-ventricular node, however, Mönckeberg's case and the one to be described below appear to be the only examples on record.

REPORT OF CASE

Abstract of clinical history. J. H. H. Out-Patient Dept. No. H82912. Mary H., a married colored woman of 39, occupied as a wool-sorter in a factory, came to the out-patient department of the Johns Hopkins Hospital on September 6, 1927, complaining of sores on her leg which refused to heal. Her family history was irrelevant. Her past history was essentially negative save for an operation eight years previously,—presumably a hysterectomy,—for the purpose of removing a "tumor from her side." She had not menstruated since then and had never been pregnant. She had had no cardiac symptoms.

Four or five months before entry, without history of trauma, a bump appeared on her right leg, followed by several others. These ulcerated but were entirely

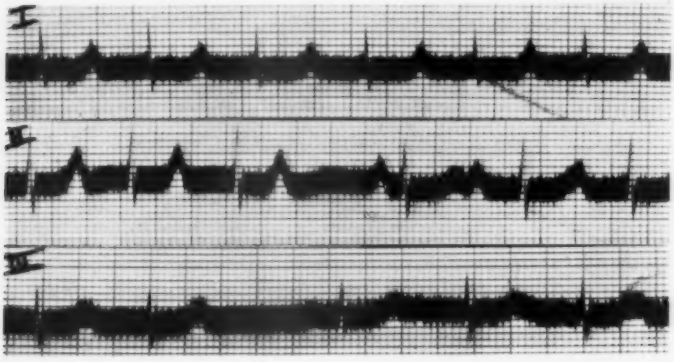

FIG. 1. ELECTROCARDIOGRAM. RATE 100. SINO-AURICULAR RHYTHM. P-R INTERVAL 0.17 TO 0.28 SECOND. FIRST DEGREE HEART BLOCK. PROLONGED CONDUCTION TIME

painless. They failed to respond to treatment with salves. She considered herself in good health otherwise and had worked every day.

Examination revealed an under-nourished colored woman with equal, active pupils and normal reflexes. The lungs were considered normal. The heart was not enlarged to percussion. On auscultation there was a rhythmical abnormality consisting of a strong cardiac contraction followed by a weaker contraction, then a pause. In each cycle the first and second sounds were definitely made out. A faint systolic murmur was heard only in the fourth intercostal space just to the left of the sternum. The blood pressure was 150/100. The pulse showed a definite bigeminal quality, with a rate of 70 per minute. The abdomen was normal save for an old operative scar in the midline between the umbilicus and pubis. The skin over the calf and anterior aspect of the right leg presented ten

or twelve small, round, punched-out ulcers, 5 to 15 mm. in diameter, some of which were healed, showing an area of central depigmentation surrounded by a pigmented areola. Others were covered with a thin brownish crust. All were superficial. A few small nodules were found on the left thigh, but no ulcers. The lesions were thought to be typical nodular syphilides of the tertiary stage.

A teleoroentgenogram showed slight enlargement of the heart and aorta. M.R. 4.2 cm. M.L. 7.5 cm. Thorax 24.5 cm.

An electrocardiogram made September 8, 1927, revealed a first degree heart block with prolonged conduction time. The rate was 100; the bigeminal rhythm was not apparent. Unfortunately the blood Wassermann test was not performed.

The patient was given three weekly intramuscular injections of bismuth salicylate in oil, and saturated potassium iodide solution (10 drops t.i.d.) by mouth. Her cardiac condition remained unaltered. On her fourth visit to the clinic she was being examined by a physician when she suddenly had a generalized tonic convulsion and died within a few minutes, without response to injections of adrenalin. While it was believed that the cardiac disturbance was in all probability due to a syphilitic lesion of the myocardium, the suggestion was made that heart block due to syphilis is usually complete and that in this case of partial block some other cause might be responsible.

Clinical diagnosis. Syphilis, tertiary cutaneous; nodular syphilide. Syphilis of myocardium; first degree heart block. Syphilis of aorta.

Anatomical findings. The autopsy was performed 24 hours after death. Only the essential portions of the protocol are here recorded.

The body is that of an undernourished adult colored female. There is no jaundice or edema. The cutaneous lesions on the right leg and left thigh are as described clinically. An old well-healed operative scar extends in the midline from just below the umbilicus to the pubis.

The pleural cavities present a few old apical adhesions. The lungs are air-containing throughout save for a small clump of old encapsulated caseous tuberculous nodules near the apex of the right lung.

The heart is not enlarged, and weighs 280 grams. The epicardium is normal. The left ventricle is distinctly dilated. The valves are delicate throughout, but the endocardium of the left ventricle is white and thickened and the trabeculae somewhat flattened. Two of the aortic cusps are partially fused at their point of insertion and one sinus of Valsalva is quite markedly dilated. No definitely syphilitic lesions are seen, however. The myocardium presents a few minute pale flecks but no obvious scars.

Thus, on routine gross examination no evidence of tumor is discovered, nor is there any obvious alteration in the contour of the inter-ventricular septum. A horizontal section through the membranous portion of the septum, however, discloses a peculiar change in the region of the atrio-ventricular node. The septum here is definitely thickened and presents a vaguely outlined cluster of minute cystic spaces surrounded by a whitish translucent fibrous stroma. The spaces

vary in size, the largest measuring scarcely 2 mm. in diameter. Some of them contain a clear yellowish coagulum. This nodule of spongy tissue has no definite border, but merges imperceptibly with the surrounding heart muscle. It measures roughly 15 by 12 by 8 mm. and lies directly beneath the endocardium of the left ventricle immediately below the center of the posterior aortic cusp, and extends through the septum to lie beneath the endocardium of the right auricle about 1 cm. below the lower edge of the foramen ovale. Section through the region of the sino-auricular node reveals no change, nor are any abnormalities seen in the muscular septum.

The remaining organs, including the brain, present no significant changes. The aorta shows no lesions characteristic of syphilis. The ovaries, tubes and fundus of the uterus have been removed. There are a few old fibrous peritoneal adhesions.

Microscopical findings. A horizontal block was cut through the membranous septum of the heart, and from this serial sections were made 10 micra in thickness. Every fifth section was stained. In brief, these show a mass of atypical tissue, definitely neoplastic in character and identical with the tumor described by Mönckeberg, invading the region of the atrio-ventricular node, which contains a few scattered bundles of muscle fibres rather smaller than those of the general myocardium. The tumor nodule is made up of ramifying spaces lined by several irregular layers of cells most of which are round or slightly elongated, with indefinite cell boundaries. Those of the deeper layers have large oval vesicular nuclei frequently with a prominent nucleolus, while those of the superficial layer are scale-like and tend to have flattened deep-blue-staining nuclei. The latter bear a close resemblance to normal endothelial cells. The larger spaces contain a pinkish coagulum showing a few scattered lymphocytes, and are separated by a dense fairly cellular connective tissue. In some areas clumps of round or oval tumor cells with vesicular nuclei are arranged in solid cords, which on examination of numerous serial sections fail to reveal any tubular structure. The tumor tissue penetrates for a short distance between the fibres of the surrounding small muscle bundles, but in general remains quite sharply localized. No definite relation can be made out between the ramifying spaces and the blood vessels.

The region of the sino-auricular node is normal. The ventricular myocardium presents a few minute accumulations of lymphocytes which have no characteristic arrangement.

Several sections of the aorta show no evidence of syphilis.

The skin lesions are of doubtful nature. The epidermis is drawn out in a smooth layer without papillary arrangement. There is a good deal of scar tissue in the corium. All the blood vessels and even the glands and crevices of the tissue are surrounded by masses of mononuclear cells, often with nodular structures which resemble tubercles, inasmuch as they have giant cells, but which are not convincingly of tuberculous origin. No tubercle bacilli or spirochetes are found in specially stained sections.

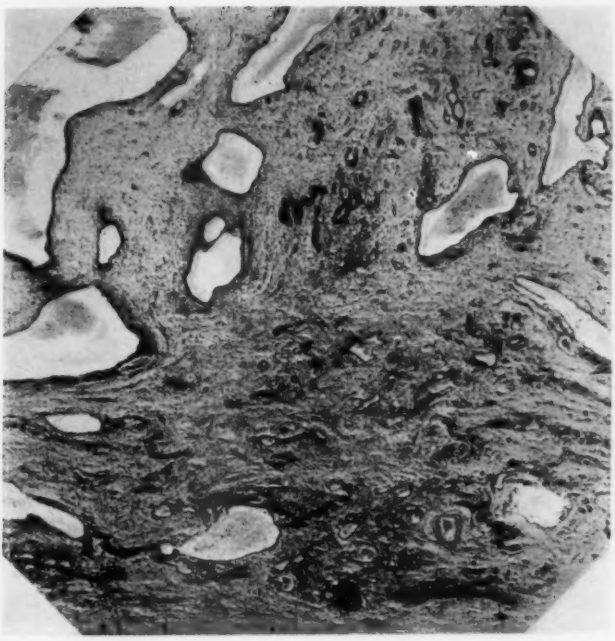

FIG. 2. TUMOR OF ATRIOVENTRICULAR NODE. 48 MM. OBJECTIVE

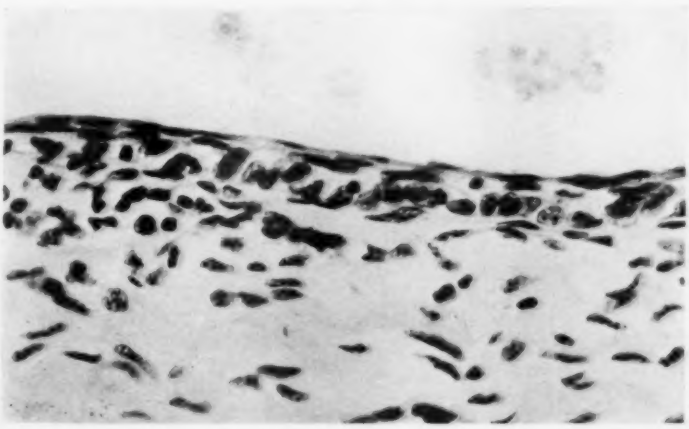

FIG. 3. WALL OF LYMPH SPACE IN TUMOR. 3 MM. OBJECTIVE

The lung presents a small cluster of old scarred tuberculous nodules with caseous centers, but no fresh tuberculous lesions are seen.

The other organs show no noteworthy changes.

The explanation of the origin of such a tumor in the atrio-ventricular node is not entirely clear. Mönckeberg states that the node is especially rich in lymphatics and believes that the tumor arises from a proliferation of their endothelium, forming a branching network of dilated lymph spaces lined by endothelium, interspersed with solid cords of endothelial cells and showing a tendency to follow the blood vessels supplying the area. The tumor reported here presents exactly this architectural arrangement. It is believed, therefore, to be a lymphangio-endothelioma, and to offer a definite anatomical explanation for the clinical findings of heart-block and sudden death. The fact that Mönckeberg's case was in a five and one-half year old boy suggests that both tumors may be congenital in origin, similar to the countless congenital angio-endotheliomata found in other locations.

SUMMARY

A case is described in which a lymphangio-endothelioma had invaded the atrio-ventricular node and the bundle of His, and caused a partial heart-block, terminating in sudden death.

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